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EYE, EAR, NOSE, AND THROAT.

A MANUAL FOR STUDENTS AND PRACTITIONERS.

BY

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PREFACE.

NEARLY all the affections of the Eye, Ear, Nose, and Throat are more or less briefly described in this volume, the more common being treated with a fulness commensurate with their importance.

Descriptive anatomy of the various regions has been omitted; but in its place are numerous anatomical figures with appropriate legends.

The section on the Diseases of the Eye is intended especially for medical students and the general practitioner. Hence, tor the sake of the *former*, nomenclature and classification have been made, it is hoped, much less complex than is the case in the ordinary text-book; while for the sake of the *latter*, those diseases of the eye for which the general practitioner is expected to prescribe are fully dwelt upon, especially those the proper diagnosis of which will lead to recognition of general diseases. The purely technical aspect of refractive errors, etc., is necessarily limited, but has been given in all its essential points.

In the section on the Ear the *physiologic tests of hearing* are given considerable prominence, as the author believes them to be of great value in the diagnosis of the lesions of the Middle Ear and Labyrinth.

Suppurative diseases of the *middle ear* and *mastoid* are described fully, with especial reference to the pathology of chronic suppuration, caries, and necrosis.

The *mastoid operation* is dwelt upon with marked detail, each step being given in the order usually employed by the author, who trusts, therefore, that this section will interest the experienced aurist as well as the student and practitioner.

Ear diseases affecting longevity are freely discussed, especially suppurative middle-ear disease, which is so commonly overlooked.

As to the Nose: The physiology and physics of *obstructed nasal respiration* as an etiological factor in many nasal and nasopharyngeal diseases are fully described.

The *rationale* of the *symptoms of sinusitis* of the accessory nasal sinuses as dependent on their peculiar topical anatomy and relation to the turbinate bodies is explained.

In the section on the Throat: Under *postnasal adenoids* there is an explanation of the symptoms which the author believes has not been given before, but which, he trusts, will, if not entirely satisfactory, at least lead to further investigation.

The etiologic *interrelationship* of chronic inflammation of the nose, nasopharynx, and larynx is strongly advocated.

The authors have made free reference to nearly all of the recent text-books of English and American authorship, as well as to a number of French and German works.

The section on the *Eye* is written by Dr. Wippern; the sections on *Ear*, *Nose*, and *Throat* by Dr. Ballenger.

WM. L. BALLENGER, M. D.
A. G. WIPPERN, M. D.

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THE EYE.

EXAMINATION OF PATIENTS.

GENERAL CONSIDERATIONS.

THE **examination of a patient** with eye trouble should be preceded by the history of the case. Traumatism, exposure to cold, foreign bodies, disturbance of vision, injudicious use of a poultice, and the coexistence of general diseases are often points of value in making a diagnosis.

Inspection of the individual will at times suggest the possible cause of the ocular trouble. Jaundice, pregnancy, or chronic alcoholism might explain poor vision at night. A waxy color of the skin and œdema of the lids might suggest Bright's disease; which in its turn would explain the disturbance of vision due to a retinitis; as would also the existence of pregnancy. The gait of a tabetic person would suggest a cause of optic atrophy. Inability to close an eye (lagophthalmos), next to exposure to cold, is due to ear disease involving the facial nerve. Ptosis with a paralytic squint in young adults generally means syphilis. The lesions of *acquired syphilis* explain the existence of paralyses, retinitis, and iritis. The evidences of *hereditary syphilis,* as notched teeth, scars at the angles of the mouth, and peculiar shape of the cranium, explain the cause of corneal trouble. The presence of a large quantity of mucopurulent secretion in the conjunctival sac signifies conjunctival disease. Photophobia occurring in children most often implies corneal disease. The broad face, concave nose, and large nostrils suggest the possibility of atrophic nasal disease and the reason for stricture of the nasal duct. A small, thick nose with an eczema of the introitus is often accompanied by conjunctival or corneal disease. Anæmia, scrofula, rheumatism, diabetes, gout, and similar diseases that depress the vital powers, may cause ocular affections which should be regarded simply as expressions of such general disease.

The diseases of the eye may be either **organic**, in which case there is a *lesion*; or **functional**, when simply an error of refraction, as hypermetropia, myopia, or astigmatism, is present.

The organic affections of the eye and appendages which can be diagnosed by **simple inspection** yield to treatment, as a rule, better than those which require the use of an **ophthalmoscope**, since they are more accessible for the application of treatment. Hence diseases of the lids, cornea, conjunctiva, iris, and lens are more amenable to treatment than affections of the choroid, retina, and optic nerve.

The correction of **refractive errors** constitutes a large part of eye-work, and is very satisfactory; but in order that this be properly done it is often necessary to understand the significance of pathological changes which interfere with the refraction and perception of light.

INSTRUMENTS.

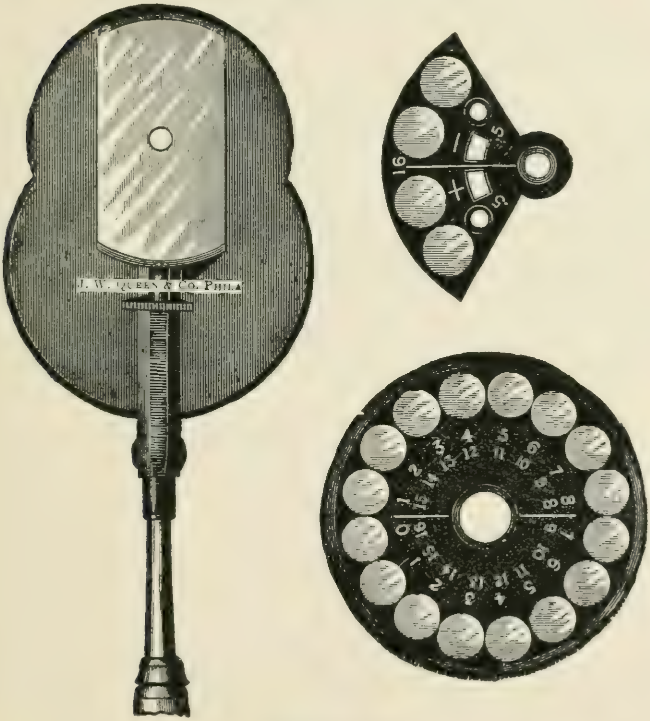
An **ophthalmoscope**, a **biconvex lens** of about 16 D., known as an object-lens, and a complete **trial-case** with *test-types* and *astigmatic chart* are absolutely indispensable for the examination of the eyes. A **retinoscope**, **perimeter**, and **ophthalmometer** are very valuable adjuncts. A host of other instruments have been devised, most of which are superfluous.

The Ophthalmoscope.

An **ophthalmoscope** (Fig. 1) is an instrument containing a more or less concave perforated mirror and a number of convex and concave lenses of different strengths, so arranged that they may be brought, preferably one at a time, behind the aperture of the mirror. Perhaps the most complete and best constructed ophthalmoscope is that made by Curry & Paxton, of London, after the pattern of Morton. The arrangement of the lenses and the presence of two concave and one plane mirror make it a superior instrument. A much cheaper instrument, after the Loring pattern, is, however, just as good for ordinary cases, and is even preferred by some. A *biconvex lens* of 16 D. is almost a part of an ophthalmoscope.

The **application** of the ophthalmoscope is a wide one. Not only can intraocular diseases be recognized and the location of opacities be determined, but the refractive condition of the eye can also be estimated. The pathological changes observed in the eye often assist in diagnosticating cerebral, renal, and general disease. The recognition of most intraocular pathological conditions is not very difficult, but the

Fig. 1.



Ophthalmoscope.

determination of refractive errors will be found very unsatisfactory unless the examining person has had great experience and is in practice. Fortunately, glasses need not be prescribed after an ophthalmoscopic examination, now that retinoscopy can so satisfactorily be applied; retinoscopy is an objective test and requires little skill.

Ophthalmoscopic examination should take place in a darkened room, and the source of light is preferably an Argand burner.

The **method** of ophthalmoscopic examination is either direct or indirect.

To examine a patient by the **direct method,** the light should be on a level with the patient's ear and on the side of the eye to be examined. The observer reflects the light into the patient's eye, using his right eye and standing or sitting at the patient's right side to examine the patient's right eye; and using his left eye and standing or sitting at the left of the patient to examine the left eye. The mirror of the ophthalmoscope should be as close as possible to the patient's eye.

The *image* obtained by the direct method is *erect,* and is magnified about 16 times regardless of the distance between the patient's and the observer's eye.

To examine a patient by the **indirect method,** the light and position should be the same as in the direct, but a biconvex lens of 16 D. should be placed at about 2 inches in front of the eye to be examined, the ophthalmoscope being about 18 inches away. This method gives an *inverted image* of the fundus and magnifies, if a biconvex lens of 16 D. be used, the image 4 times.

The ophthalmoscopic examination should be preceded by one with the aid of **oblique or focal illumination**: the biconvex lens of 16 D. is so held that the rays of light from the lamp, which should be at the side and slightly in front of the patient, are condensed upon the part to be examined.

To examine the *cornea,* the glass should be held farther from the eye than when an examination of the *lens* is undertaken. By an examination with oblique illumination, opacities of the cornea and the anterior portion of the lens, the relative depth of the anterior chamber, iridodonesis, and changes of the iris can be easily recognized. Changes in the peripheral and posterior parts of the lens, and even in the anterior portion of the vitreous, can be observed when the pupil is dilated. An examination by oblique illumination will explain a disturbance of vision from opacities otherwise overlooked, and will save in such cases much time and trouble.

The ophthalmoscopic examination of an eye can often be satisfactorily made without the use of a **mydriatic**; but in some cases, more numerous with beginners, dilatation of the pupil will be found necessary.

To examine the **refracting media**, the ophthalmoscope should be held about 8 inches from the eye examined, the observer being emmetropic. For a myope the distance should be less; and a hyperope or presbyope would require a lens of + 3 or + 4 D. or more in the aperture. The opacity can not only be seen, but, by observing the parallactic displacement of the opacity when fixed with reference to the margin of the pupil, its location determined.

An *opacity in the cornea* seems to move in a direction opposite to the movement of the mirror; one upon or near the anterior capsule remains stationary in all positions of the mirror; and one near the posterior capsule seems to move in the same direction.

In order to examine the cornea or lens *very carefully*, a convex lens of 16 to 20 D. should be brought into the aperture.

The refraction of the eye may be determined with the ophthalmoscope by either the direct or the indirect method, but the former is almost exclusively employed.

When both the observer's and the patient's eye are emmetropic, the ciliary muscle being relaxed, the details of the fundus can be distinctly seen through the aperture.

When the patient is myopic and the observer emmetropic, the concave lens necessary to show the details of the fundus represents the degree of myopia.

In the same way, the degree of hypermetropia is determined by the strength of the convex lens necessary to show the fundus distinctly.

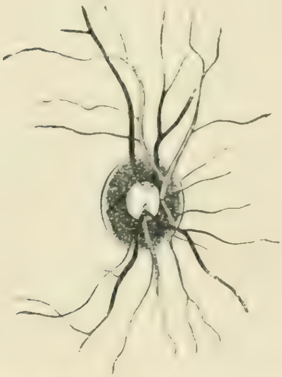
When the observer is either hypermetropic or myopic the same rule applies; but the observer must have corrected his refractive error.

If the observer will notice the *movement of the vessels,* whether it is with or against the movement of the mirror, ametropia can be inferred: when there is hypermetropia the vessels move "with;" when myopia, "against."

Astigmatism is demonstrated in an eye by the direct method (Fig. 2), when the optic disk is seen elongated in the direction of the meridian of greatest refraction. The retinal vessels are most distinctly seen in the meridian of the greatest ametropia.

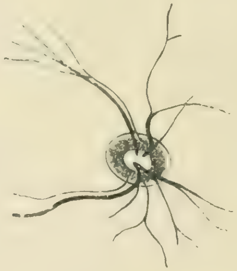
When the *indirect method* (Fig. 3) is employed, the eye being emmetropic and the accommodation relaxed, the image of the disk remains the same in size and shape for every distance of the biconvex lens from the eye. With an increase of this distance, in hypermetropia, the image becomes smaller; and in myopia larger; when no astigmatism is present the original shape is always retained. When astigmatism is present, recession of the lens causes variation in both size and shape of the image of the disk. The elongation of the disk occurs at right angles to the meridian of greatest refraction, provided the lens is at a distance from the eye less than its focal distance. At its focal distance from the eye the disk

FIG. 2.



Erect image of disk in astigmatism, with meridian of greatest refraction nearly vertical. (de Wecker and Jäger.)

FIG. 3.



The same disk as shown in Fig. 2, seen by the indirect method. (de Wecker and Jäger.)

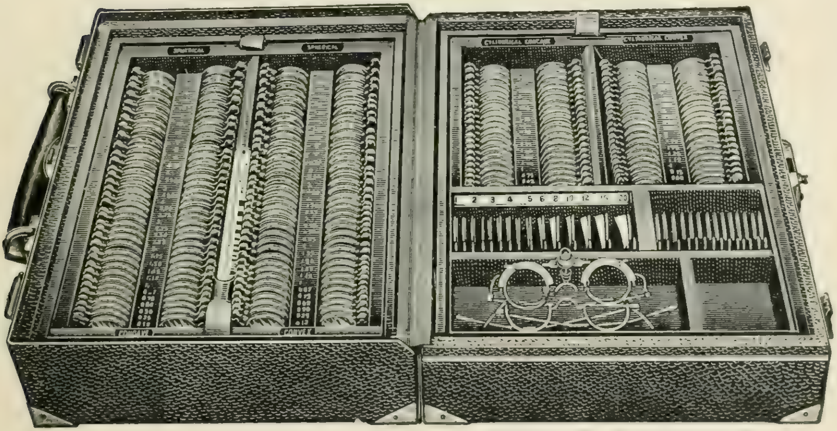
would appear circular; and beyond that distance would seem elongated at right angles to the long axis of the image when the lens is within its focal distance from the eye.

The direct and indirect methods may be employed to determine *differences of level in the fundus;* but the former method is almost exclusively employed. A difference of refraction of two points in the fundus amounting to 3 D. means $\frac{1}{25}$ inch difference of level. In cases of papillitis with a swollen disk or glaucoma with cupping, this method may be employed to measure respectively the amount of swelling or cupping.

Trial-case. Test-types. Astigmatic Chart.

The **trial-case, test-types,** and **astigmatic chart** (Figs. 4, 5, 6), like the ophthalmoscope, are indispensable, and for the correction of refractive errors constitute the best resource. With a complete trial-case supplied with prisms, the relative strength of the extraocular muscles and the degree of the heterophorias, and even of strabismus, can be ascertained.

Fig. 4.



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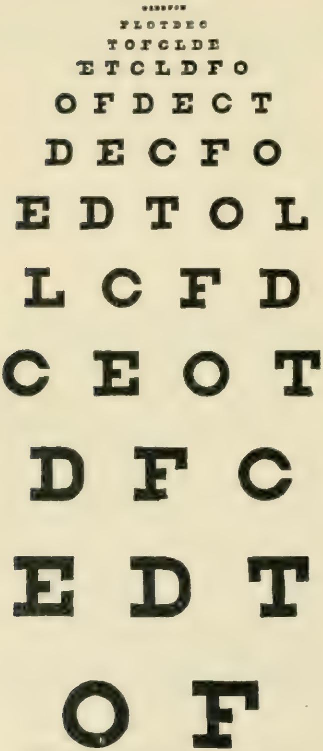
Trial-case.

The test-types, including a chart of illiterate distance-type and one of test-type for near tests, are of value to determine the visual acuity, with or without glasses. In order to **test the acuity**, a space 20 feet in length should be available, since at that distance the rays which come to a focus upon the retina are parallel and the accommodation is not required. The visual acuity of most cases should be determined and recorded in order to discover disturbance of vision. The *test-types* are arranged in rows which, according to the size of the letters, can by a normal eye be read at specified distances. When the row marked 20 can be read at 20 feet the vision is written $\frac{20}{20}$, and is normal. When the row marked 120 is the smallest that can be seen at 20 feet the vision is written $\frac{20}{120}$. When all the letters but three can be read in the row marked 40 at 20 feet the vision is expressed $\frac{20}{40} - 3$. The *illiterate distance-type* is of value in the examination of both children and adults who cannot read the Roman letters. Such patients who have sufficient intelligence can with their fingers show whether or not they see the direction of the arms of the figures utilized. When not sufficiently intelligent for even this form of examination, the patients, either small children or foreigners, should (after ophthalmoscopic examination) be subjected to a retinoscopic examination and fitted accordingly.

FIG. 5.



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Test-types.

A complete trial-case should contain the following:

30 pairs each of convex and concave spherical lenses from 0.25 D. to 20 D.

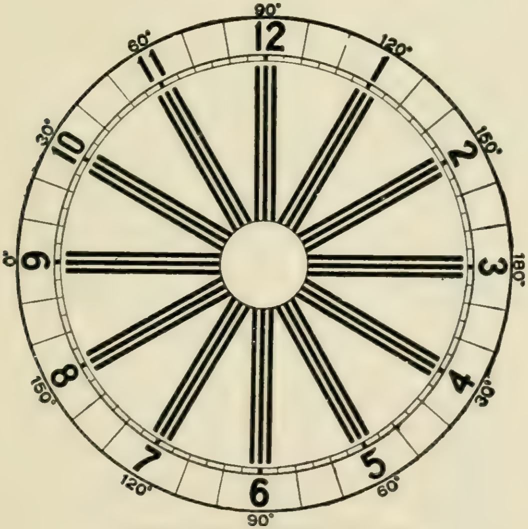
20 pairs each of convex and concave cylindrical lenses from 0.25 D. to 6 D.

16 prisms, from ½ degree to 20 degrees.

1 plano blue and 1 plano red glass.

6 plano smoked glasses, 6 shades.

FIG. 6.



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Astigmatic chart.

1 solid disk of rubber and 1 ground-glass disk.

1 pin-hole disk.

1 stereoptic disk.

1 Maddox rod.

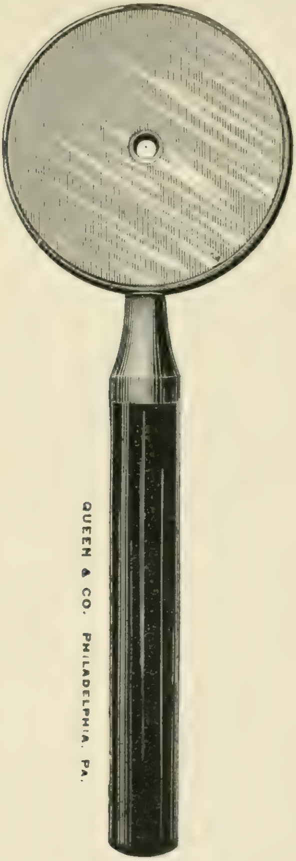
1 Maddox double prism (improved).

1 graduated trial-frame.

1 plain trial-frame.

The Retinoscope.

Fig. 7.



Retinoscope.

The **retinoscope** (Fig. 7) is the most useful instrument for the objective determination of refractive errors. It is nothing more than a plane or concave mirror perforated at the centre and supplied with a handle. The Thorington retinoscope has a plane mirror and can be recommended.

Retinoscopy is the method of determining the refraction by observing the change of illumination of the pupil that occurs when the mirror of the retinoscope is rotated on its vertical and horizontal axes. When the mirror is turned on its vertical axis, the refraction of the horizontal meridian is determined; and when on its horizontal axis, the refraction of the vertical meridian may be computed. In order *to practise retinoscopy* a dark room and a single source of light, as an Argand burner, are necessary. The *light* may be placed directly over the patient's head; or, when Thorington's method is employed, the burner, which should be covered with a chimney in which there is a round aperture of about one-half inch diameter, is placed near the head of the examining person, who reflects the light from the aperture to the patient's eye. The *distance* between the patient and the examined person may vary, 20, 40, or 80 inches being the distance employed to suit the case. When the *mirror is turned* on its vertical axis, it will be noticed that the edge of the shadow is usually vertical, and that the shadow moves horizontally across the pupil either in the direction of the movement of the mirror or against it. In the former case the shadow is said to move "with," in the latter "against."

When a *plane mirror* is used, the distance of the mirror from the examined eye being about 40 inches, the rules are as follows :

The mirror rotated → :

←θ— Shadow "against" implies myopia more than 1 D.	—θ→ Shadow "with" implies myopia less than 1 D., emmetropia, or hypermetropia.
A series of concave lenses should be placed before the examined eye, the other being covered, beginning with a weak one. The last concave lens which causes the shadow to go "against," increased by 1 D., is the refraction.	A series of convex lenses should be placed before the examined eye, the other being covered, beginning with a weak one. The first convex glass which causes the shadow to move "against," diminished by 1 D., is the refraction.
When the shadow moves quickly it indicates a low degree of myopia ; when slowly, a high degree.	When the shadow moves quickly it indicates emmetropia, myopia less than 1 D. ; or a low degree of hypermetropia.

When a *concave mirror* is used, the rules for the movements of the shadows given above are reversed. When the distance between the mirror and the examined eye is 20 inches, then 2 D. are either subtracted or added to the finding, as the case may be. When the distance is 80 inches, or 2 metres, 0.5 D. is either added or subtracted.

In an *emmetropic*, or normal, eye the shadow will be reversed by placing before it a convex lens of 1 D., if the distance between the mirror and the examined eye is the focal distance of a glass of 1 D.—that is, 40 inches. Hence the rays of light emerging from the cornea were parallel before the lens was used.

Suppose it be found that a + 3 D. is the weakest convex lens necessary to cause reversal of the shadow (the distance between the mirror and the eye being 40 inches) in both the vertical and horizontal meridians, then the condition may be formulated thus : $-\Big|\overset{+3}{\text{—}} + 3$. After subtracting 1 D., the refractive condition would be expressed thus : $\overset{+2}{-\!\!\!|\!\!\!-}\ +2$. Since a convex lens of 2 D. causes normal refraction in both meridians, a convex spherical lens satisfies the condition.

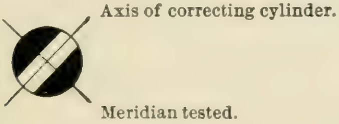
Suppose a + 3 D. reverses the shadow in the vertical meridian and a + 4 D. is necessary in the horizontal meridian, then the refractive condition of the eye may be expressed thus : $\overset{+2}{-\!\!\!|\!\!\!-}\ +3$. The correcting glass is a convex spherical lens of 2 D. (which corrects the vertical meridian and 2 D. of the horizontal) combined with a cylinder of 1 D. (the difference between 3 and 2), its axis being at right angles to the meridian to be corrected—hence vertical or at 90 degrees. The glass may then be expressed + 2 sph. ⊃ + 1 cyl. × 90 or + 2 sph. ⊃ + 1 cyl. axis vertical. This case is one of compound hypermetropic astigmatism.

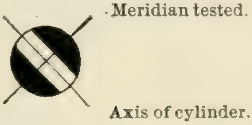
In a case of *simple myopia* of 3 D. it will be found that a − 2 D. is the strongest concave glass which does not reverse the shadow in both meridians. When a − 4.5 D. is the strongest concave glass which does not reverse the shadow in the vertical meridian and a − 3 D. the strongest glass for the horizontal meridian the refraction would be expressed : $\overset{-5}{-\!\!\!|\!\!\!-}\ -4$. The correction would be − 4 sph. ⊃ − 1.5 cyl. × 180 for this case of compound myopic astigmatism.

Suppose a − 3 D. is the strongest concave glass that does not reverse the shadow in the vertical meridian, and a + 3 D. is the weakest convex glass that reverses the shadow in the horizontal meridian, then the refraction would be expressed thus : $\overset{-4}{-\!\!\!|\!\!\!-}\ +2$. This is a case of mixed astigmatism. The correcting glass might be designated in three ways : − 4 cyl. × 180 and + 2 cyl. × 90 ; − 4 sph. ⊃ + 6 cyl. × 90 ; or + 2 sph. ⊃ − 6 cyl. × 180. The last-mentioned way is preferred by the optician, who likes working − cylinders on to + spheres.

In converting two cylinders into a sphere and cylinder this fact should be kept in mind : a sphere is equal to two cylinders of the same kind and degree when the axes are at right angles to each other: $+ 2 \text{ sph.} = \begin{cases} + 2 \text{ cyl.} \times 180 \\ + 2 \text{ cyl.} \times 90 \end{cases}$. Take the example above: − 4 cyl. × 180 combined with + 2 cyl. × 90. By using a + 2 sph. in place of a + 2 cyl. × 90, the glass is too strong by a + 2 cyl. × 180. Therefore it is necessary to subtract a + 2 cyl. × 180, which is the same as adding a − 2 cyl. × 180 to − 4 cyl. × 180, which makes a − 6 cyl. × 180.

When the shadow *moves horizontally* from side to side the edge of the shadow is vertical, but the horizontal meridian is the one tested. A cylinder to correct an error must be placed at right angles to the meridian requiring correction. Since the edge of the shadow is at right angles to the meridian tested, the position of the edge of the shadow represents the axis of the cylinder required for correction. Hence, by noting the position of the edge of the shadow the axis of the correcting cylinder is determined:





Occasionally the axis of the correcting cylinder will be other than horizontal or vertical, and the retinoscopic examination will often determine the proper axis.

The Perimeter.

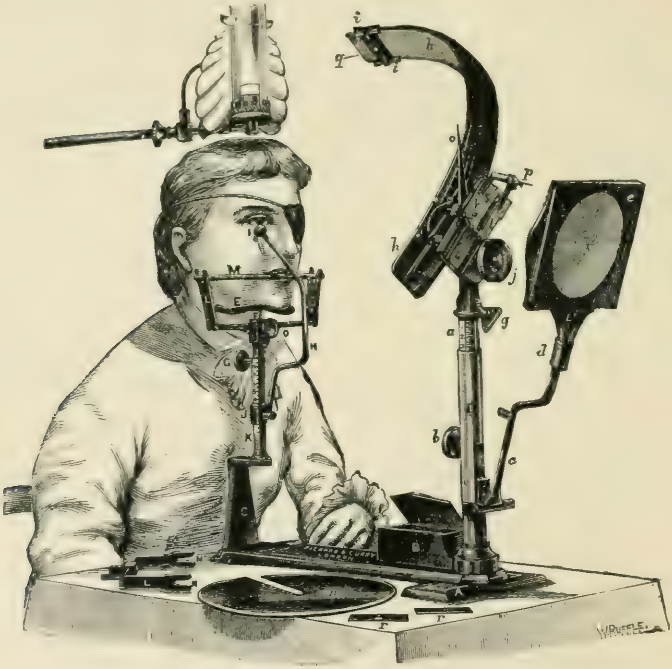
The **perimeter** (Fig. 8) is an instrument devised to determine the field of vision.

The **field of vision** can be determined in a general way by the hand, and better yet by a graduated blackboard; but the use of a perimeter is the most accurate and scientific method.

To determine the field of vision with the *hand*, the patient should stand with his back to the window and the examiner should have his eyes on a level with those of the patient. When the right field is to be determined, the patient should be directed to look continually at the examiner's left eye. The examiner then moves his left hand in various positions, and in a relative way the size of the field of vision can be determined.

When a *blackboard* is used, this should be divided into meridians and concentric circles by lines, and the patient is directed to fix his eye at the centre. The chalk is then advanced on the blackboard at the greatest distance within the field of view, and this line gives the field of vision. The various parts of the retina are measured at different distances by this method, which is for this reason inferior to **perimetric examination**:

FIG. 8.



McHardy perimeter.

The *perimeter* is a semicircular band of metal which is divided into degrees, beginning at its middle. It turns upon an axis at the middle point in such a way that it passes through all meridians. A slide is moved along the arc so that concentric circles can be made within 90 degrees.

The *patient* is seated with his chin in an adjustable rest in such a position that the examined eye is directly in front of the middle of the arc. A card made specially for the right or the left eye is placed at the back of the axis of the arc and turns with the arc. The card is divided into meridians and concentric circles, and is so placed that movements of the arc correspond with the meridians on the card. The point nearest the periphery at which the patient can see the slide on a given meridian is noted on the card, and these points are ascertained for all the meridians. A line connecting these points determines the visual field. Circumscribed areas which cannot be seen in the visual field are called *scotomata.*

A **scotoma** may be central, peripheral, or annular, and may exist for only certain colors. Normally the fields of vision for various colors are not the same, and the examination of the visual field to determine the relative visual fields for colors is of value in the diagnosis of disease. A diminution of blue means disease of the retina ; of red and green, the optic nerve (Fig. 9).

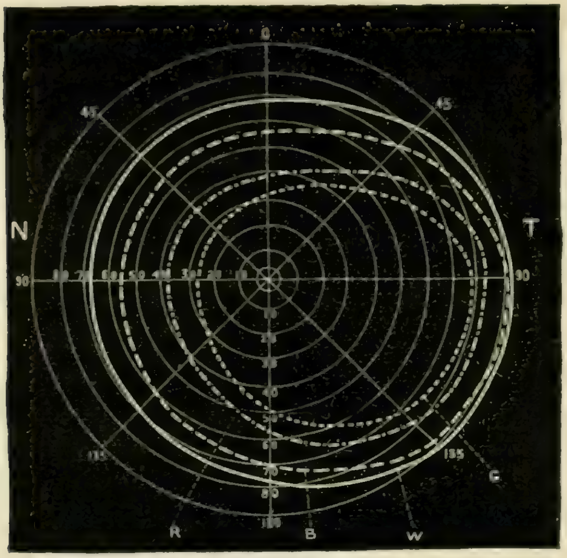
In making a perimetric examination, it will be observed that a **blind spot** is always present to the temporal side of the point of fixation between 10 and 20 degrees. This is due to the optic disk, which is not sensitive.

Hemiopia, which implies that one lateral half only of the retina is functioning, can be easily demonstrated with the perimeter. When the left halves of the visual field of both eyes are intact the hemiopia is said to be *homonymous.* The same applies to the right side. *Temporal hemiopia* implies that only the temporal side of the retina of both eyes is functioning, and hence the visual field is intact on the nasal sides. Homonymous hemiopia occurs from a lesion in either the right or left optic tract. Temporal hemiopia is the result of a lesion near the middle of the optic chiasm.

When the visual field is narrowed at the periphery so that the limits of the visual field are equally diminished the condition is called **concentric contraction.** This condition is often observed in retinitis pigmentosa and occasionally in glaucoma.

Scotomata occur frequently in choroidal or retinal disease when the lesion is circumscribed.

Fig. 9.



Field of vision of right eye as projected by the patient on the inner surface of a hemisphere, the pole of which forms the object of regard (half-diagrammatic). T, temporal, N, nasal side. W, boundary for white; B, for blue; R, for red; G, for green. (Landolt.)

The Ophthalmometer.

The **ophthalmometer** (Figs. 10, 11) is an instrument for measuring the corneal curvature of the eye, and for determining the amount and character of corneal astigmatism by noting the variations in that curvature. It consists mainly of two bright, or luminous, objects (mires) whose images are reflected by the corneal surface; of a telescope through which the images are seen enlarged; and a birefringent prism within the

FIG. 10.

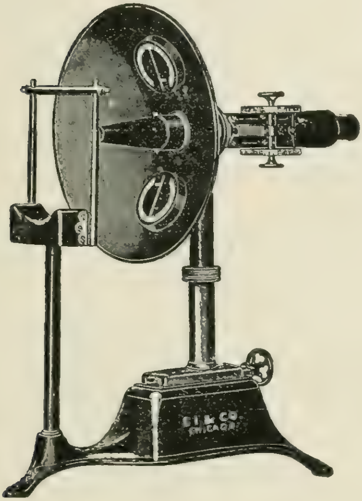
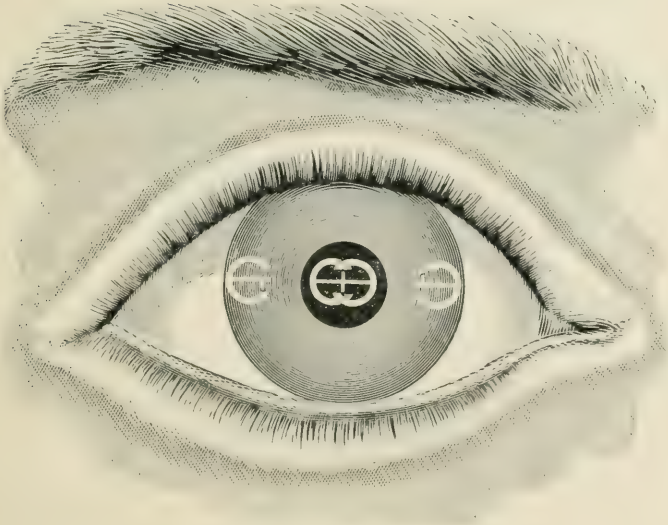


FIG. 11.



tube of the telescope, which brings the images into apparent proximity. Each mire has a long central line which is in exact alignment with the corresponding line on the other mire; and a shorter line, or spur, at right angles to the larger one, which may be brought to appear in alignment with its fellow on the other mire by moving the prism within the tube. The prism is provided with a rack, and is moved forward or backward in the tube by means of a pinion which carries a graduated scale.

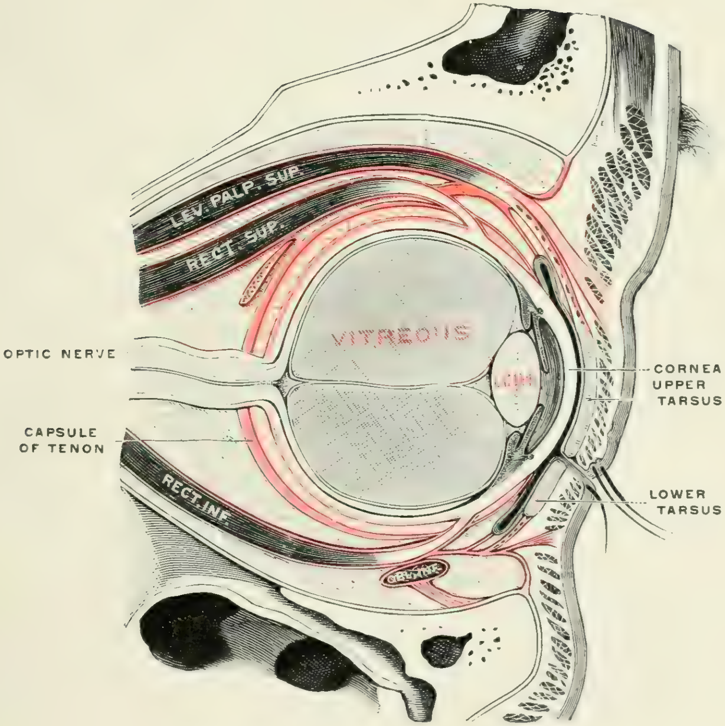
Each cornea examined, acting as a graduated reflector, shows between the spurs of the images an amount of space inversely proportional to its diminishing power—that is, its curvature. The prism is moved nearer the eye or farther from it until this distance between the spurs is exactly eliminated. The scale attached to the pinion will then indicate the curvature in diopters. The entire apparatus as described may be rotated before the eye to be examined and the curvature measured at various meridians. The longer lines of the mires will appear in exact alignment upon an astigmatic cornea only when they are in the meridians of greatest or least curvature—that is, at the axes. The difference between the curvature at the two axes is the amount of corneal astigmatism.

It is evident that the ophthalmometer affords a rapid and objective method to determine the amount and axis of corneal astigmatism, and that its intelligent use will often save time and trouble in correcting errors of refraction. Ophthalmometers of different construction are on the market, yet the one described and illustrated, made by Chambers Innskeep & Co., is superior to any foreign or domestic instrument with which we are familiar.

THE ORBIT.

Anatomy: The orbits (Plate I.) are two cavities which contain the organs of vision. Seven bones, the frontal, sphenoid, ethmoid, superior maxillary, malar, lachrymal, and palate, enter into the formation of each orbit. The bony walls of the orbits have their periosteum, and next to this, mesially, is more or less fatty cellular tissue, which forms a bed of support for the globe. Next comes the capsule of Tenon, which is "serous" membrane consisting of a visceral and a parietal

PLATE I.



The Right Eye in Sagittal Section, showing the Capsule of Tenon. Semi-diagrammatic. (Testut.)

layer. The parietal layer clothes the cavity of the fatty tissue in which the globe is imbedded; and the visceral layer covers the posterior part of the globe from near the margin of the cornea to the entrance of the optic nerve. The ocular muscles near their insertion pierce the capsule of Tenon, which gives off some fibres to reinforce the sheaths of the muscles. The orbit contains periosteum, fatty cellular tissue, the lachrymal gland, the capsule of Tenon, the ocular muscles, nerves, bloodvessels, and bulb.

Malformations: In acephalous monsters, the anterior opening of the orbits is directed upward; in double-faced monsters with three or four eyes, the orbits are not fully developed; and in cases of cyclopia, the single common orbital cavity is of small size and the optic foramina are at times wanting. In cases of microphthalmos and anophthalmos the orbit is in part defective. When loss of the eyeball occurs in infancy, the orbital cavity does not always attain the mature size. In such a case, a rubber ball or wax cast should, if possible, be worn to supplant the bulb, in order to encourage the development of the orbit.

Neoplasms: Tumors of the orbit may arise in the bony walls and periosteum, the optic nerve, or in the cellular tissue. They produce exophthalmos, strabismus, and more or less loss of motility.

The bony walls of the orbit may be the seat of osteomas. These are usually very hard, and their removal is often possible only after enucleation of the eyeball. The danger from meningitis and cerebral abscess is great in removal of osteomas of the roof of the orbit.

The cellular tissue of the orbit may be the seat of cystoid and dermoid tumors, angiomas, lipomas, and sarcomas.

The early removal should be thoroughly performed in all cases of orbital tumors.

Injuries of the Orbit.

The **margin** of the orbit, because of its prominence, is subject to injury from blows or falls.

Contusions and *incised* or *lacerated wounds* often occur in the soft parts overlying the margin of the orbit, and usually cause little trouble.

Fracture of the malar bone may cause anaesthesia from injury of the infraorbital nerve and diplopia from injury of the inferior oblique muscle. Some injuries involve both the supraorbital and infraorbital nerves.

Fractures of the orbital walls produce such *hemorrhage* that there are usually exophthalmos and ecchymosis of the lids and conjunctiva. When a fracture of the orbital wall extends to the optic foramen the sight may be lost.

Gunshot wounds may produce immediate blindness from injury to the optic nerve or rupture of the choroid, although the eyeball appears uninjured.

When the lachrymal bone can be easily displaced from the orbital plate of the ethmoid and when hemorrhage from the nose occurs, emphysema of the orbital tissues being present or not, there is a fracture of the *inner orbital wall.*

Emphysema of the orbital tissues may be produced by fractures causing a communication between the orbit and the antrum of Highmore, the frontal or sphenoidal sinuses, the ethmoidal cells, or the nasal duct.

Injuries, muscle operations, and foreign bodies may cause orbital abscesses. These should be opened by exploratory punctures, and relief will be given even though no pus be found.

Fractures of the orbital walls should be *treated* according to the rules of surgery. Detached pieces of bone must be removed; and should an abscess develop, this is to be thoroughly drained. Emphysema of the orbital tissue usually terminates favorably with avoidance of sneezing and coughing and application of a light bandage.

Diseases of the Orbit.

Orbital cellulitis is an acute, subacute, or chronic inflammation of the fatty cellular tissue, and may be idiopathic or traumatic in origin.

When *acute* the disease presents a formidable appearance. The pain in the orbit is excruciating. There are fever, headache, œdema of the lids, chemosis of the ocular conjunctivæ, exophthalmos, neuroretinitis, ulceration and suppuration of the cornea, and panophthalmitis. When due to erysipelas, the most severe symptoms are likely to develop. Thrombosis of the orbital veins and cavernous sinus may occur in the course of an orbital cellulitis. The *treatment* depends upon the severity of the symptoms. Numerous incisions through the conjunctiva, or through the lids if it be impossible to open them, should be made to relieve the tension and secure drainage. Irrigation of the wound with bichloride of mercury solution (1 : 2000) should be made from time to time. Hot applications hasten the process. If complicated by abscess of the cornea and panophthalmitis, evisceration of the bulb may be required. Enucleation is not so safe as evisceration in these cases, since cerebral complications are more likely to occur after the former.

Periostitis is due to injury, cold, foreign bodies, or syphilis. When *acute*, there are slight fever, great pain, tenderness on pressure, and the lids are red and swollen. Caries or necrosis of the underlying bone may result after a time, which can be ascertained by probing the fistulous opening which usually occurs through the lids.

In *treating* this condition the fistula should be enlarged if necessary, and the dead bone removed. The wound should be kept clean and open to favor healthy closure from the bottom. Acute periostitis should be treated as an orbital cellulitis by puncture and drainage.

When *due to syphilis*, the pain is greater at night. For these cases antisyphilitic treatment should be instituted.

Foreign bodies causing periostitis should be removed.

Tenonitis, or inflammation of the capsule of Tenon, may be of idiopathic or traumatic origin.

The upper lid is swollen and œdematous, and movement of the eyeball is painful. Exophthalmos when present is slight.

The *idiopathic* form is often due to rheumatism or gout.

Hot fomentations to the lids and antirheumatics and anodynes usually bring about recovery.

In the *traumatic* form, antiseptic solutions should be used to cleanse the conjunctival sac; and free incisions should be made as soon as the formation of pus is evident.

Exophthalmic goitre: Basedow's or Graves's disease causes more or less exophthalmos and draws attention to the eyes. Enlargement of the thyroid gland, tachycardia, and protrusion of the eyeball are the cardinal symptoms of the disease, which is primarily an affection of the nervous system.

When the cornea suffers from inability to close the lids (lagophthalmos), it may be necessary to use a bandage or perform a tarsorrhaphy, whereby the lids are united only to such an extent as not to interfere with vision.

Pulsating exophthalmos presents the following symptoms: protrusion of the eyeball, noises audible over the orbit, and pulsation felt on the eyeball and about the orbit. It may be spontaneous or traumatic. Most of these cases are due to rupture of the internal carotid artery in the cavernous sinus.

The *treatment* is compression of the common carotid artery; and if done without result ligation is indicated.

THE EYELIDS.

Anatomy.

The eyelids (Fig. 12) cover and protect the eyeball.

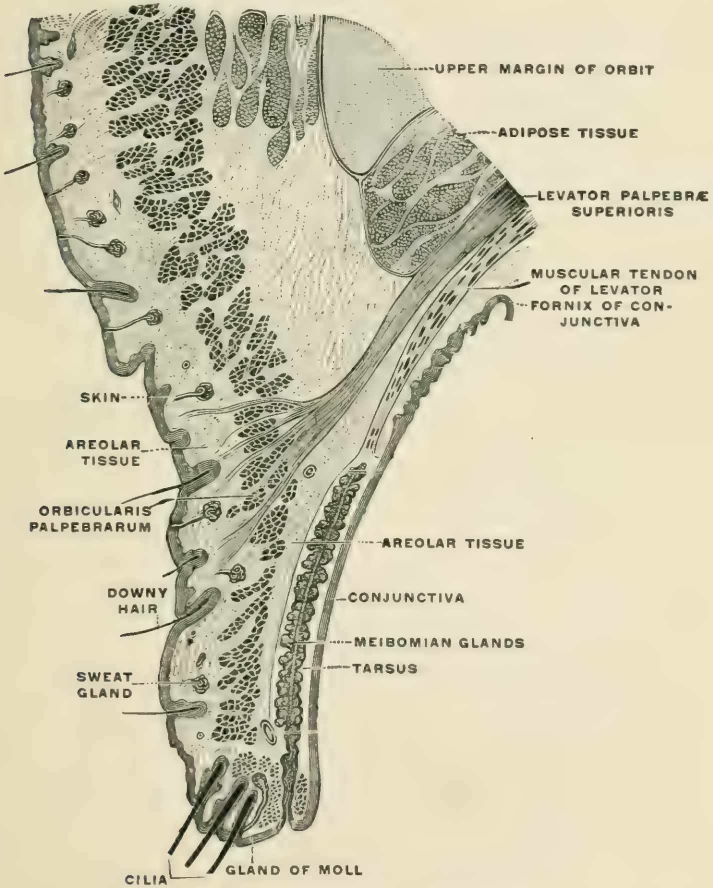
The **palpebral fissure** is the space between the free margins of the lids; the outer angle is known as the *outer canthus*, the inner angle as the *inner canthus*.

The **upper lid** is larger than the lower, and has a special muscle to raise it, the *levator palpebræ superioris*. The free margin of the lid has an outer and an inner lip. The *outer lip* is rounded and contains two or three rows of hairs, known as the *cilia*. The inner, or posterior, lip contains the mouths of the *Meibomian glands*; and near the inner canthus the *lachrymal papilla*, at the summit of which is the *punctum lachrymale*.

From without inward, the lids are composed of integument, subcutaneous areolar tissue, muscles, fibrous tissue (known as *tarsal cartilage*), Meibomian glands, and conjunctiva.

The **integument**, which is studded with fine, downy hairs, is very thin, and because of the loose connection with the areolar tissue is lax.

Fig. 12.



Upper lid in sagittal section. (After Merkel.)

The **subcutaneous areolar** tissue (containing no fat) connects the integument and muscle. It contains **hair-follicles** and **sweat-glands.**

The following **muscles** are in the lids: the palpebral portion of the orbicularis palpebrarum, the ciliary muscle of Riolanus, the tensor tarsi, the aponeurosis of the levator palpebræ superioris, and the superior and inferior palpebral muscles.

The **orbicularis palpebrarum** is a sphincter muscle, and consists of two portions: an orbicular, acting for the most part involuntarily; and a palpebral, subject to the will. When both portions act together, the lids are brought into close contact, as occurs in photophobia. The palpebral portion is concerned in gently closing the eyes. It is supplied by the facial nerve.

The *muscle of Riolanus* is that small part of the orbicularis palpebrarum between the Meibomian glands and the follicles of the cilia.

The *tensor tarsi*, or *Horner's muscle*, is that portion of the orbicularis palpebrarum situated at the inner side of the orbit arising from the crest of the lachrymal bone and adjoining surface of the orbit. It draws the eyelid inward, and by compressing the punctum lachrymale on the globe facilitates the drainage of tears.

The **levator palpebræ superioris** is the antagonist of the orbicularis palpebrarum, for it raises the eyelid. It arises near the upper margin of the optic foramen, and is inserted by a broad aponeurosis into the anterior surface of the tarsal cartilage. It is supplied by the third nerve.

The **superior palpebral** is a small involuntary muscle composed of non-striated muscle-fibres. It arises from between the fibres of the levator palpebræ superioris just before it becomes aponeurotic, and is inserted into the under surface of the upper margin of the tarsal cartilage.

The **inferior palpebral** muscle arises from the orbital connective tissue.

These muscles are supposed to bring the free margins of the lids in perfect apposition when the eyes are closed. They are supplied by the sympathetic.

The **tarsal cartilages** are two plates of dense fibrous connective tissue. The superior is of a semilunar form measuring about one-half inch at its broadest part and tapering toward the extremities. The *lower* smaller and thinner tarsal cartilage is elliptical in form. It measures one-quarter inch at its broadest part. The outer and inner extremities of each cartilage are held to the margin of the orbit by the outer and inner *canthal ligaments.* The inner canthal ligament is known as the *tendo-palpebrarum.*

The **Meibomian glands** are imbedded in grooves in the inner surface of the tarsal cartilages. There are from 30 to 35 in the upper lid and from 20 to 30 in the lower lid. They correspond in length with the breadth of the tarsal cartilages, and hence those of the upper lid are the longer. A Meibomian gland consists of a straight tube with a blind extremity, into which small secondary follicles open. These sebaceous glands supply a secretion which prevents adhesion of the lids. The infant has very large Meibomian glands.

The outer lip of the free margin of the lids has protruding from it two or three rows of hairs, the **cilia.** Those of the upper lid curve upward and are longer than those of the lower lid, which curve downward.

Small **sebaceous follicles** (the glands of Zeiss), and modified sweat-glands (known as Moll's glands), are among the roots of the cilia which lie in the connective tissue between the orbicularis palpebrarum and the tarsal cartilage. One of Zeiss's glands opens into each hair-follicle, and the secretion serves to lubricate the cilium. The mouths of Moll's glands open just behind the cilia.

Malformations and Congenital Anomalies.

Coloboma is a fissure of the lid ; it may be congenital or traumatic. The upper lid is more often the seat of the congenital form. It is sometimes associated with dermoid growths of the cornea or with colobomas of the iris and choroid. *Treatment* consists in freshening the edges and stitching them together.

Epicanthus is a malformation in which a crescentic fold of skin overlaps the inner canthus. This condition is often associated with ptosis and strabismus.

The overlapping skin may be excised ; or the operation of cutting out an oval piece of skin from the bridge of the nose, the long axis being vertical, and closing the wound by sutures improves the condition.

Ectropion, entropion, ptosis, distichiasis, ablephoria (partial or total), **blepharophimosis, symblepharon, anklyloblepharon,** and **cryptophthalmos** may be congenital.

Neoplasms (Benign).

Xanthelasma, or *xanthoma*, has been classified with neoplasms; but recent researches have shown that it is the product of the degeneration of embryonically misplaced muscle-fibres.

Xanthelasma *occurs* as yellowish-white patches seldom raised above the skin. This condition is most often found after middle age, particularly in females. The patches generally occur on the upper lid, near the inner canthus.

The *treatment*, usually desired for cosmetic effect, is excision.

Molluscum contagiosum is a small round tumor affecting the sebaceous glands. It may attain the size of a pea; the top is flattened, and in the centre is a dark spot, representing the aperture of a follicle. It is not contagious, as was once thought.

The *treatment* consists in evacuation of the cheesy mass; sometimes it is necessary to excise the walls of the cavity and apply a strong solution of silver nitrate.

Angioma: A *nævus*, or "mother's mark," is always congenital, and may cover the whole lid. When small it may be successfully *treated* by the application of caustics, such as nitric or chromic acid, or by the actual cautery.

Telangiectasis is a bright-red patch occurring in the skin and subcutaneous tissue, and consists of enlarged capillaries, arterioles, and venules.

Cavernous angioma is a vascular tumor composed of anastomosing sinuses with a framework of connective tissue.

These tumors are often encapsulated, and can then be enucleated. When extensive, electrolysis is the best treatment. Multiple punctures with the electrolytic needle are made around the base of the tumor.

Papillomas (due to hypertrophy of the papillæ of the skin) should always be removed, since they are sometimes the starting-point of epitheliomas.

Fibromas, enchondromas, myxomas, lipomas, lymphangiomas, and **neurofibromas** but rarely occur on the lids.

Neoplasms (Malignant).

Epithelioma begins on the skin usually at the border of the lid, and unless excised early invades the bulb and even the orbit. It occurs more frequently on the eyelids than elsewhere. It commences as a small inflamed elevation, whose surface becomes excoriated and secretes a watery or bloody liquid which forms a crust or scab. This falls off from time to time, showing an indurated ulcerated surface. The progress of the ulceration is very slow, and pain is not severe unless the deeper structures be involved.

When thoroughly removed at the beginning epitheliomas may not recur, but after they are large and have ulcerated an operation is not satisfactory.

Rodent ulcer is a form of epithelioma which leaves cicatricial tissue in the part invaded. The ulcers of lupus vulgaris and secondary syphilis are liable to be confounded with epithelioma.

Epithelioma is a disease of advanced age ; syphilis, of youth or middle age. Epithelioma is much slower in progress than syphilis. Lupus commences usually before puberty and is slower in progress than epithelioma.

Sarcoma of the eyelid is nearly always secondary. It is characterized by rapid growth and early recurrence. Thorough removal is the *treatment.*

Injuries of the Eyelids.

The eyelids are often the seat of **incised** or **lacerated wounds, contusions, burns,** and **scalds.**

Owing to the laxity of the skin, **œdema** and **ecchymosis** of the lids may be considerable.

As the **result of injury** either to the eyelid itself or to the orbit, producing periostitis and caries, *abscess* of the lid is not uncommon. *Ulcers* sometimes occur, the result of scalds, burns, or contusions. A *traumatic coloboma* may be the result of a vertical wound where no union by first intention takes place.

Emphysema of the lids is a symptom of fracture of one or more bones entering into the formation of the orbit. The source of the air producing this condition is from the frontal or superior maxillary sinuses, the ethmoidal cells, or the nasal cavity. When by reason of sneezing, coughing, or blowing the nose the air in the spaces mentioned is under pressure, air is forced into the areolar tissue at the seat of fracture.

Œdema may be due to congestion in the lids, the conjunctiva, or the orbit, and is often an expression of diseases of the heart and kidneys. When the swelling is excessive, puncture and application of pressure relieve the condition.

Ecchymosis is an extravasation of blood in the subcutaneous tissue, and the most common cause is contusion.

Injuries of the eyelids—treatment: The best treatment immediately after the injury is the application of cold, either ice or iced-water. In order to *promote absorption*, the application of heat and a firm bandage are recommended.

Abscess is usually the result of contusions, although spontaneous abscesses occasionally occur in scrofulous or poorly nourished children. The application of heat hastens the process and a horizontal incision is indicated to evacuate the pus.

Ulcers from whatever cause should be treated by attention to cleanliness and the application of antiseptics.

Since *emphysema* is caused largely by blowing the nose, the proper conditions being present, the avoidance of this act is the only treatment.

Wounds should be treated in accordance with the rules of surgery.

Skin Diseases.

The eyelids may be the seat of many **skin diseases**, but the consideration of a few of the more common or peculiar will suffice.

Eczema is the most common of the skin diseases. The etiology is often the same as in other parts, yet, owing to the irritation of the skin by the tears, either as the result of rubbing with the hands or conjunctivitis, lachrymal catarrh, or ectropion, the lower lid may become affected. Inflammation, itching, and moisture make the diagnosis easy.

The *treatment* varies evidently with the cause. For *simple* *cases* the application of Hebra's ointment (equal parts of lead plaster and vaseline), zinc salve, or a 5 to 10 per cent. solution of silver nitrate is the usual treatment.

When the eczema is *secondary* to other diseases the necessary treatment should be applied to them.

Erysipelas of the eyelids occurs in the course of facial erysipelas, and the superficial form is without danger.

The *phlegmonous type* may involve the capsule of Tenon and the deeper tissues of the orbit, and impair or destroy the sight from optic neuritis or atrophy. A meningitis from extension of inflammation along the optic-nerve sheath is rare and is usually fatal.

The *treatment* should consist in local applications of ichthyol, guaiacol, or 10 per cent. solution of silver nitrate and a supporting medication. The success of the treatment will depend largely upon the strength of the patient.

Herpes zoster ophthalmicus is a neuropathic disease characterized by the formation of vesicles along the course of one or more branches of the ophthalmic nerve. Neuralgia and fever may precede the eruption, which, owing to the attending redness and swelling, simulates erysipelas.

The administration of anodynes to relieve the pain and the dusting of the affected parts with zinc oxide or rice flour are indicated.

Milium is a term applied to concretions of sebaceous matter about the size of a millet-seed. Simple expression is the *treatment*. This disease may be mistaken for the commencing stage of xanthelasma palpebrarum, the distinguishing features of which are the clear yellow color, limitation to the eyelids, and the flat end plate-like arrangement of the lesions.

Spontaneous ulcers of the lid may be due to lupus, syphilis, or scrofula. The *treatment* of the ulcer of lupus consists in the use of the curette or actual cautery and the application of antiseptics. The syphilitic ulcer is usually a late manifestation of syphilis, and should be diagnosed early, since antisyphilitic treatment is effective, and surgical measures superfluous and even dangerous. The scrofulous ulcer demands constitutional as well as local treatment.

Inflammation along the ciliary margin, *blepharitis ciliaris*, appears in two forms: squamous and ulcerative.

Blepharitis squamosa is a subacute or chronic disease of the border of the eyelid characterized by the formation of scales and exudation of a tenacious secretion, often glueing the cilia together. On removal of the scales or crusts a more or less hyperæmic surface presents itself, and often several lashes are pulled out in the process. The lashes, however, usually grow again.

Blepharitis ulcerosa differs from the squamous form in that the inflammation is deeper, the secretion purulent, and the loss of cilia permanent.

Blepharitis—etiology: Anæmia, scrofula, eruptive fevers, neglect of cleanliness, exposure to dust, heat, winds, and irritants produce bilateral disease. Corneal opacities, a high degree of hypermetropia (causing strain of accommodation), and uncorrected errors of refraction are exciting causes. Chronic catarrhal conjunctivitis, phlyctenular conjunctivitis, trachoma, and epiphora are local causes. When epiphora produces blepharitis the cause should be ascertained, whether increased secretion or disease of the lachrymal canal or sac. Blepharitis may cause a chronic catarrhal conjunctivitis, a stye, loss of the cilia, tumefaction along the border of the lids (particularly the upper), epiphora from eversion or narrowing of the puncta, trichiasis, and ectropion.

Blepharitis—treatment: This depends upon the cause. When constitutional treatment is needed, the iodide of iron, cod-liver oil, fresh air, milk and egg diet, and baths are indicated. *Local applications* are useful only after thorough removal of the scales and crusts. This is best accomplished by soaking the lids in a warm alkaline solution. A weak solution of hydrozone is also effective. The lids being cleaned, the application of a salve containing 1 per cent. to 4 per cent. of the yellow oxide of mercury, or 1 per cent. of the white precipitate, effects a cure in simple cases. In the *ulcerative form*, besides the removal of crusts, the infected area should be opened and the cilia involved removed by the use of ciliary forceps. The ulcers heal quicker upon the application of a strong solution of silver nitrate.

Phthiriasis may be mistaken for blepharitis; but the eggs and the parasites may be detected by close examination. Mercurial ointment effects a cure.

Eyelids: Diseases of the Glands.

Hordeolum: An *external stye* is the result of a suppurative inflammation of one of Zeiss's glands. Swelling and redness occur over the site of the gland and increase for a few days. After spontaneous or operative evacuation of the contents all inflammatory symptoms rapidly subside.

An *internal stye* is the result of suppurative inflammation of a Meibomian gland. Inasmuch as these glands are larger than those of Zeiss and are imbedded in the dense fibres of the tarsus, an internal stye is more painful and lasts longer than an external one. On everting the lid the pus may be seen through the conjunctiva.

Etiology: Styes occur in young people, particularly the scrofulous and anæmic. Blepharitis causes a stye by the presence of scales and crusts, which serve as a bed for germs which easily enter the glands. The swelling of the margin of the lids may prevent the exit of the secretion of the glands. Errors of refraction, and strain of accommodation may be exciting causes.

Treatment: Hot applications accelerate the ripening of the abscess and opening by free incision shortens the course of the trouble. As a prophylactic measure treatment of an existing blepharitis is imperative.

Chalazion is a disease of the Meibomian glands characterized by its chronicity, and the formation of a tumor which causes comparatively little inconvenience, and finally degenerates into granulation-tissue and pus. It is easily differentiated from an internal stye, which runs an acute course, is painful, and suppurates.

Chalazia *occur* more frequently in adults than in children. They may produce some deformity and irritate the eye by reason of the chafing caused in winking.

Treatment: Small chalazia call for no treatment; large ones should be removed. The incision may be made through the skin, conjunctiva, or margin of the lid. The contents should be thoroughly scooped out with a chalazion scoop and the walls of the sac scraped or dissected out. The application of a strong solution of silver nitrate often produces adhesive inflammation of the cyst-wall. Cocainization is usually sufficient for this operation.

Cretaceous deposits in the conjunctiva sometimes cause irritation. After cocainization, they should be picked out. This condition is known as lithiasis conjunctivæ, and is due to degeneration of the contents of a Meibomian gland.

Anomalies of the Eyelashes.

Trichiasis is that condition, not accompanied by inversion of the margin of the lid, in which the cilia are entirely or in part directed inward instead of outward. The irritation of the eyeball causes photophobia and lachrymation. Corneal ulcers and opacities may also occur in consequence of mechanical injury to the cornea.

Etiology: The most common cause is trachoma, when the trichiasis is often complete—that is, involving all of the cilia. Partial trichiasis, in which only some of the cilia are directed backward, occurs as a result of scars due to blepharitis, stye, injury, or operation.

Distichiasis is a rare and generally congenital condition in which an otherwise normal lid has two rows of cilia, one directed forward, the other backward.

Treatment: When only several cilia are directed inward epilation may suffice. As this does not destroy the follicle, epilation must be repeated from time to time.

The radical treatment is the destruction of the follicle by *electrolysis*. The positive pole of a galvanic battery (eight to twenty elements) is connected with a flat electrode, the negative pole with the needle. The needle is thrust into the follicle by the side of the lash and the flat electrode is placed on the temple. This treatment is practicable only when a few of the cilia turn in. The painfulness of this method militates against its usefulness. When many or all of the cilia are directed inward, epilation cannot be practised. Here the best treatment is an operation to remedy entropion.

Anomalies of Position of the Lids.

Entropion is a turning in of a lid. It may be spasmodic or cicatricial. *Spasmodic entropion* is caused by contraction of the orbicularis muscle. Two conditions are necessary to produce this kind of entropion: imperfect support of the free border of the lid and excessive relaxation of the skin.

Etiology: The relaxation of the skin as occurring in the aged, the absence or smallness of the bulb, an eye deeply set in the orbit, blepharospasm, blepharophimosis, or the use of a bandage.

Treatment of spasmodic entropion depends upon the cause. When produced by the use of a bandage, its discontinuance is all that is necessary. If the bandage be indispensable, the inversion of the lower lid may be prevented by the use of a strip of adhesive plaster, one end of which should be stuck to the skin over the edge of the orbit. The use of an artificial eye prevents entropion when caused by absence of the bulb.

Cicatricial entropion is caused by cicatricial shortening of the conjunctiva whereby the free border of the lid is turned inward.

Etiology: Trachoma, diphtheritic conjunctivitis, and injuries of the conjunctiva.

The *operation* for the relief of entropion will be considered in the section on Operations.

Ectropion is an eversion of the lid whereby a part of the conjunctival surface is exposed. Eversion of the *puncta* occurs in consequence of the eversion of the margin of the lid, and hence lachrymation is a prominent symptom. The irritation of the secretions causes redness and thickening of the lid-margins and hypertrophy of the conjunctiva. High degrees of ectropion produce lagophthalmos, which may lead to corneal disease.

The *etiology of ectropion* is various. It may be due to spasm which everts the margin of the lid when the tarsal portion is slightly turned from the eyeball and the integument is tense. Protrusion of the eyeball favors spasmodic ectropion. Paralysis of the orbicularis, because of the relaxed condition of the lower lid, may cause paralytic ectropion. In old age the loss of tone of the orbicularis and chronic catarrh of the conjunctiva favor senile ectropion. When scars occur in the integument the result of injury, operation, burns, and the like, cicatricial ectropion results.

Treatment: In *spasmodic ectropion* the lids should be replaced and a firm bandage applied. *Paralysis of the facial nerve* requires the use of electricity. When the ectropion exists in high degree surgical means are necessary to give relief.

Symblepharon is the adhesion of the palpebral to the ocular conjunctiva in consequence of injuries causing excoriation or sloughing of the opposing surfaces. The adhesion may be complete, in which case the cul-de-sac is obliterated; or partial, when it remains free, permitting the passage of a probe under the bridge of tissue along the fornix between the lid and bulb.

Etiology: Burns, corrosion, diphtheritic conjunctivitis, operations, and ulcers of all kinds may produce symblepharon.

The object of the *treatment* is to break up the adhesions and prevent union of the exposed surfaces. This can be accomplished in cases of partial symblepharon, but is very difficult or impossible in complete ones.

Ankyloblepharon is the adhesion of the margins of the lids, the canthi not being involved. The *etiology* is the same as for symblepharon, which generally complicates this condition. Two opposing surfaces being denuded by reason of burns, etc., may become adherent.

The *treatment* consists in permanent separation of the lid-margins after a dividing cut has been made. The success of the treatment will depend upon the extent of the symblepharon when it coexists.

Lagophthalmos is a condition in which the lids cannot be completely closed. It may be due to shortness of the lids, ectropion, paralysis of the orbicularis, absence of corneal reflex, and enlargement or protrusion of the bulb.

The *treatment* depends upon the cause. When the lagophthalmos is due to shortness of the lids a canthoplasty is necessary. Lagophthalmos is relieved by an operation for ectropion when this causes it. Paralysis of the orbicularis, which is supplied by the facial nerve, may be due to a variety of causes, such as injury to the nerve, exposure to cold, or intracranial diseases. It can therefore not always be treated. When the lagophthalmos is only a temporary condition, the eye should be bandaged, to protect the cornea from injury and irritation.

When, however, the condition is not likely to yield to treatment a *tarsorrhaphy* should be performed, to prevent certain destruction of the eyeball.

Blepharophimosis is the condition in which the palpebral fissure is shortened, either as a result of contraction of the lids or adhesion of the opposing margins of the lids at the outer canthus. A *canthoplasty* is the treatment.

Diseases of the Muscles of the Lids.

Spasm and *paralysis* of the orbicularis frequently occur.

Spasm, or **blepharospasm**, as it is called, may be a symptom of many ocular diseases in which there is irritation. Refractive errors, foreign bodies in the conjunctival sac, trichiasis, or whatever disease may be the cause, should receive attention. Blepharospasm occurs also as a disease of itself. It is observed in hysterical young women, and also in old age, when the lids may be quite closed or continually winking. When attempts are made to raise the upper lid there is considerable resistance, which differentiates this trouble from ptosis. When of hysterical origin blepharospasm often passes away without treatment. In the aged treatment is of no use.

Paralysis of the orbicularis is due to a lesion of the facial nerve; this is more often peripheral, seldom central. Exposure to cold, rheumatism, traumatism, disease of the middle ear or temporal bone, neoplasms, and syphilis are the most common *causes*.

The electric current is of use in all forms, in conjunction with the special *treatment* that may be indicated. The eye should be always bandaged, to protect the cornea, in severe cases; at night only in simple cases. When there is no prospect of recovery a tarsorrhaphy should be made to close the lids partially.

Ptosis is a paralysis of the levator pelpebræ superioris. It may be congenital, when it is usually bilateral; or acquired, when it is usually unilateral. The upper lid droops more or less, interfering with the sight when the margin of the lid is below the level of the pupil. Lesions involving the muscle or the third nerve are the causes of acquired ptosis. It is often associated with paralysis of the ocular muscles supplied by the third nerve. Ptosis is sometimes seen in the paralytic form of alcoholism.

The *treatment* depends upon the cause. In some cases surgical procedure is the only means of help.

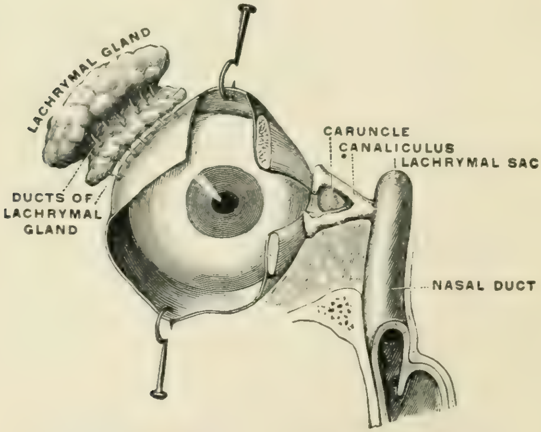
THE LACHRYMAL ARPARATUS.

Anatomy.

The lachrymal gland, the accessory lachrymal glands, and the tubular acinous glands of the conjunctiva constitute the **secreting part** of the lachrymal apparatus; the lachrymal canals, the lachrymal sac, and the nasal duct, the **conducting part.**

The **lachrymal gland,** which secretes the tears, is lodged in the lachrymal fossa under cover of the external angular process. It is divided into two parts, a larger (or upper), and a lower, the *accessory gland.* The secretions of the upper gland pass through the ducts of the accessory gland and finally empty into from seven to twelve narrow ducts which pierce the outer side of the superior fornix of the conjunctiva.

FIG. 13.



The lachrymal apparatus of the right eye. (Testut.)

Only a small amount of the tears is secreted by the lachrymal and accessory glands, the greater part coming from the *tubular acinous glands* of the conjunctiva. The lachrymal gland can accordingly be extirpated without evil consequences, the conjunctival glands supplying secretions sufficient to moisten the surface of the eyeball. The lachrymal gland is supplied by the lachrymal artery, a branch of the ophthalmic. The lachrymal nerve, a branch of the fifth, and branches of the sympathetic supply the gland.

The **lachrymal canals** collect the tears when excreted to excess. These begin at the puncta lachrymalia, which are opposite each other on the free border of the eyelids near the inner canthus, turning toward the eyeball, and lead to the **lachrymal sac.** This lies in a groove at the lower inner angle of the orbit, and gradually merges into the **nasal duct,** which opens into the inferior meatus about one-half inch behind the anterior end of the inferior turbinal.

The lachrymal, superior maxillary, and inferior turbinated bones form the *bony canal* of the nasal duct, which is lined, from within outward, by mucous membrane, a layer of veins, and a fibrous sheath. Temporary engorgement of the veins may interfere with the passage of a probe. When the lachrymal canals and nasal duct cannot accommodate the excess of tears these flow over the lower lid and down the cheek.

Congenital Malformations.

The lachrymal glands may be **absent,** when there is usually other malformation, as anophthalmos or cryptophthalmos. When children do not shed tears the lachrymal gland may be present, but the *tubular glands* of the conjunctiva are probably absent.

Dislocation of the lachrymal gland is sometimes congenital, and **fistulæ** have also been observed.

The lachrymal canals sometimes **fail to unite,** when a groove instead of a canal results.

At times the **canaliculi** are but **partially closed**; or they may be wanting.

Atresia of the puncta may be congenital.

A **fistula** of the lachrymal sac or of the nasal duct is a rare occurrence, but **stricture** of the latter is more frequently observed.

Neoplasms.

Tumors of the **lachrymal gland** are rare. Adenomas, myxomas, myxosarcomas, sarcomas, epitheliomas, cysts, dermoid tumors, and angiomas have had their origin in the gland.

The **removal** of the gland should be performed early when it is possible to save the eye. When the growth has involved too much tissue, **enucleation** is imperative.

Polyps have been met with in the **lachrymal canal.** They should be removed.

Injuries.

Injuries about the orbit sometimes produce a **fistula of the lachrymal gland,** allowing the tears to escape through the opening in the integument. The fistula should be brought into communication with the conjunctival sac, after which the integument may be freshened and the external opening closed.

Dislocation of the lachrymal gland may be due to traumatism or may occur spontaneously.

Wounds involving the puncta, the lachrymal canals, and the nasal duct may produce **atresia** of any or all of these parts. The possibility of restoring the function depends upon the nature of the injury.

For *atresia of the punctum,* a dilator should be frequently used after the punctum has been opened. If the *canaliculi* are closed, it is necessary to open them with a canaliculus knife and pass probes.

In cases of traumatic stricture of the *nasal duct* it may be necessary to make an opening with a knife or drill, under general anæsthesia, after which the probe should be frequently used. If this fails, a gold canula is of great service.

Diseases.

On the whole, disease of the lachrymal gland is very rare; disease of the drainage or conducting apparatus, on the other hand, is quite common.

Dacryo-adenitis, or inflammation of the lachrymal gland, is a very rare disease. For an acute inflammation the application of heat and an incision as soon as fluctuation is demonstrable are indicated. Antisyphilitic or antirheumatic remedies are useful in the treatment of the chronic form in conjunction with local stimulating applications.

Dacryops, or cyst of the lachrymal gland, is the result of closure of one or more of the excretory ducts, whereby the retention and accumulation of secretion produce a transparent elastic swelling. The *treatment* consists in establishing a communication between the cyst and conjunctival sac.

Chalky concretions sometimes form in the lachrymal gland. As soon as they cause irritation they should be removed.

Lachrymal Blennorrhœa.

Definition: This is a chronic disease of the mucous membrane of the canaliculi, the lachrymal sac, and nasal duct.

Symptoms: Because of the swelling of this mucous membrane the drainage of the tears is interfered with, and accordingly *epiphora*, or overflow of tears, results. In light cases this may be the only symptom.

As the disease progresses a *stricture* occurs in the drainage-apparatus, in consequence of which the lachrymal sac increases in size from retention of secretion. This becomes infected, and soon the lachrymal sac on pressure empties through the canaliculi a mucopurulent secretion.

Etiology: Uncorrected errors of refraction may cause simple cases. Exposure to cold, causing a conjunctivitis or rhinitis, is also an etiological factor in some cases. Hypertrophic or atrophic rhinitis, syphilitic or tuberculous ulcers and tumors, and polyps in particular, are nasal conditions which may lead to stenosis and inflammation of the drainage-apparatus.

Complications: Lachrymal blennorrhœa often causes chronic conjunctivitis from irritation of the mucous membrane by the purulent secretion, and ulcerating blepharitis from overflow of tears. Finally this disease leads to eczema, shortening of the lid, and entropion.

The **treatment** depends upon the cause. When due to syphilis, the appropriate treatment not only relieves lachrymal disease, but prevents caries and necrosis of the lachrymal bone.

Nasal diseases should receive attention when they are causative. In all cases the patient should be instructed to press out frequently the purulent secretion; and cleanliness is to be enjoined. In some cases the passage of the probe accomplishes a cure in a few months; but very often the physician is not justified in promising much.

For passage of the probe, the punctum must be large enough to permit its introduction into the canaliculus, which must also in turn be large enough to allow the passage of the probe into the sac. The punctum can be dilated with a pointed probe; the canaliculus must sometimes be cut before using the probe. For this purpose a canaliculus-knife is pushed through the canaliculus till the resistance of the lachrymal bone is felt, when the cutting edge of the knife should be brought upward and inward.

The lachrymal syringe is of use in most cases, but should never be used immediately after any cutting operation, for fear of producing infiltration of the tissues. A saturated solution of boric acid or a 1 : 5000 solution of bichloride is generally used in the lachrymal syringe. After such a solution has been used for a time it is well to substitute a weak solution of silver nitrate, 1 to 5 grains to the ounce, or zinc sulphate of the same strength.

Dacryocystitis occurs during the course of a lachrymal blennorrhœa. The lachrymal sac becomes the seat of a violent inflammation, which manifests itself by pain, redness, and swelling of the lids. The constitutional reaction is at times great, so that the disease may be confounded with facial erysipelas by the inexperienced. If left alone, a spontaneous opening, known as a *lachrymal fistula*, results.

If seen at the beginning, the passage of a probe and antiseptic injections may abort the disease; but when a lachrymal abscess is imminent it is better to encourage its formation by hot applications. When ripe an incision should be made to evacuate the pus. So long as the lachrymal fistula remains the danger of a new attack of dacryocystitis is *nil.* After the inflammatory signs have subsided, the drainage-apparatus should receive attention, and the fistula should not be allowed to close so long as there is a stricture in the nasal duct. If the nasal duct cannot be kept open, the lachrymal sac should be excised. This prevents a recurrence of the dacryocystitis; and as removal of the lachrymal sac removes a source of irritation, the lachrymation is much diminished.

The destruction of the lachrymal sac should be undertaken in syphilitic or strumous patients when there is caries of the lachrymal bone.

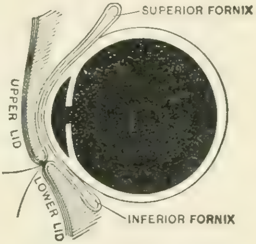
THE CONJUNCTIVA.

Anatomy.

The **mucous membrane** forming the inner surface of the lids and the covering of the anterior half of the bulb from the equator to the corneal margin, is the **conjunctiva** (Fig. 14).

The **limbus** is that part of the conjunctiva which marks the corneal margin where the epithelial layer of the conjunctiva becomes transparent and begins to form the anterior layer of the cornea.

FIG. 14.



Sagittal section of eye, showing superior and inferior fornices of the conjunctiva. (Testut.)

The conjunctiva consists of palpebral and ocular parts, which are connected by intervening folds, each known as the fornix.

The **palpebral conjunctiva** begins at the free margin of the lid, forming a continuation of its integument. It is also continuous with the lining membrane of the Meibomian glands. Dense cellular tissue firmly unites the tarsal cartilage and underlying conjunctiva, which, because of its thinness, makes it easy to see the outline of the Meibomian glands. Disease of the nasal mucous membrane sometimes affects the conjunctiva, since the lining membrane of the lachrymal canaliculi and sac leading to the nasal duct is a continuation of the conjunctiva.

The **ocular conjunctiva** is that part loosely connected to the bulb by connective tissue. It is very thin, is freely movable over the bulb, and contains no glands and few papillæ.

The crescentic fold of conjunctiva at the inner canthus is the **plica semilunaris**, in which rests partly the **caruncula lachrymalis**. This contains various glands which secrete a whitish substance frequently seen at the inner canthus.

A **cul-de-sac** is formed in the upper and lower lids by the reflection of the corresponding **fornix**. The *superior cul-de-sac* is the deeper, but is difficult to examine without a double eversion of the upper lid. The *lower* is easily seen on requesting the patient to look upward, the lower lid being at the same time held down.

The **fornix** is the thickest part of the conjunctiva, and because of its rich blood-supply and laxity of structure takes on great swelling in inflammations of the conjunctiva. The fornix contains lymphatic follicles and accessory lachrymal glands (Krause's).

The *superior* cul-de-sac contains numerous tubular acinous glands, the secretion of which is similar to that of the lachrymal gland. It is from this source that the eye is constantly kept moist, the lachrymal gland functioning only at times, such as in crying. The presence of the lachrymal gland is accordingly not very important.

The fornix, because of its elasticity and folds, permits the needful motility of the bulb.

Conjunctiva—function: It forms the smooth inner surface of the lid, and its glands furnish secretions which lubricate the parts, thus facilitating movements of the lids and eyeball without friction, and which moisten the cornea, thus preserving its clearness and integrity.

Congenital Abnormalities of Conjunctiva.

Subconjunctival fatty growths, nævi, tumors composed of bone-tissue, and dermoid tumors may be congenitally present in the conjunctiva.

Dermoid tumors most frequently occur, and are often associated with **colobomas** of the iris and the choroid. The sclerocorneal junction is the usual seat of these growths, and the cornea is more or less involved.

When, for cosmetic reasons or inconvenience, the patient seeks help, the *treatment* is removal.

Neoplasms.

Granulomas occur in the wake of healing wounds and injuries of the conjunctiva. Foreign bodies imbedded in the cul-de-sac frequently cause them.

A **polypus** resembles a granuloma, which can be distinguished from it by the absence of the epithelial covering which the polypus always possesses. Polypi usually occur on the caruncle.

Papillomas often spring from the caruncle, but may also be found in the palpebral conjunctiva.

Treatment: *Granulomas, polypi,* and *papillomas* should be removed and the stumps cauterized when broad.

Besides these, cysts, angiomas, fibromas, myxomas, and osteomas occur in the conjunctiva.

Epitheliomas and **sarcomas** are the malignant growths which involve the conjunctiva. They occur in old age, and unless radically removed in the earlier stages the patient succumbs.. These growths usually spring from the limbus of the conjunctiva and spread over both cornea and conjunctiva.

Treatment: When these malignant growths cannot be thoroughly removed by use of the knife, curette, and cautery, the eye should be *enucleated,* even though vision may not be affected.

Injuries.

Foreign bodies: Dust, cinders, ashes, and small particles of similar substances often lodge on the eyeball, and, on winking, are carried away, only to become again lodged in the palpebral conjunctiva of the upper lid, usually in the subtarsal fold. The *pain*, which is sometimes very severe, is due to the chafing of the cornea by the foreign body during winking.

Treatment: On everting the lid the foreign body is easily removed. Great mischief is sometimes done by the patient, who, at the suggestion of friends, applies raw steak or poultices of various kinds, producing serious infection.

Foreign bodies of *considerable size* occasionally lodge in the fornix, and may remain there for some time without inconvenience.

Wounds of the conjunctiva are usually attended with ecchymosis. They should be *treated* on surgical principles.

Corrosion and **burns** of the conjunctiva often occur as the result of contact of acids, caustic alkalies (lime), steam, hot water, molten metal, and the like.

Symptoms: Not only is there loss of conjunctival tissue, but also frequently *symblepharon*, which more or less interferes with the motility of the eye. The impairment of sight depends upon the severity of the injury to the cornea.

The **treatment** consists in removal of the corrosive substance as soon as possible, and attempts to neutralize or render it inert. When injury is due to *caustic alkalies*, milk instead of water should be used to flush the conjunctival sac. Injury due to *lime* is best treated first with application of oil and then with instillations of a saturated solution of cane-sugar.

The *inflammations* produced by burns and corrosion are best treated with cold applications, atropine, and the bandage.

In *severe injuries* the lids should frequently be lifted from the bulb, in order to prevent adhesion of the denuded surfaces.

When *symblepharon* does occur, an operation must be done to relieve to some extent the adhesions which prevent motility of the bulb.

DISEASES OF THE CONJUNCTIVA.

Acute Catarrhal Conjunctivitis.

Definition: This is an inflammation of the conjunctiva lasting in uncomplicated cases from one to two weeks.

Its **extent** varies with the severity of the disease, simple cases involving the palpebral conjunctiva and fornix ; severer ones, the conjunctiva of the bulb as well. There is hyperæmia of the mucous membrane and the secretion of mucus is increased.

Symptoms: The patient complains of photophobia and the sensation of a foreign body in the conjunctival sac, which is produced by the presence of stringy mucus. Burning and itching of the lids occur, but there is no great pain unless the cornea and iris be involved. The secretions dry upon the edges of the lid during the night, gluing the lids together in the morning.

Etiology: Exposure to cold is the most common cause. Many cases occur in the spring-time, when the disease may be epidemic. The bacillus of Weeks is concerned in the etiology when the proper conditions are present.

Treatment: *Simple cases* of acute catarrhal conjunctivitis require only attention to cleanliness. A saturated solution of boric acid flushed into the conjunctival sac every two or three hours is all that is necessary. Simple cases are sometimes transformed into complicated ones by injudicious treatment. Mydriatics and cocaine should not be instilled; and bandages, poultices, and compresses never be used. Eye-strain predisposes to conjunctival catarrh, and errors of refraction should receive attention in all cases apparently not infective.

Severer cases are best treated by applications of a 2 per cent. silver nitrate solution, care being taken that the solution does not come in contact with the cornea. This treatment should be carried out daily, besides attention to cleanliness. If the patient cannot be seen daily, a solution of zinc sulphate or silver nitrate (0.25 per cent.) may be prescribed for home-use. Several drops of this are to be instilled three times daily. Hydrozone neutralized with borax in solution (1 grain to the ounce), and diluted to suit the tolerance of the case (about 1 : 10) makes an excellent antiseptic application. In cases of infective origin, this treatment, together with frequent cleansing of the conjunctival sac, is all that is necessary.

Chronic Catarrhal Conjunctivitis.

This is an affection of adults, particularly in old age.

Symptoms: The patient complains of various sensations, such as dryness, burning, lachrymation, and fatigue of the eyes, particularly in evenings; and it will be noticed that the conjunctiva is slightly congested, the lid-margins thickened in consequence of blepharitis, and a small amount of tenacious secretion accumulates at the inner canthus. The secretion often glues the lids together during the night.

Treatment of this very frequent disease demands attention to errors of refraction, the general health, and regulation, if possible, of the employment, that irritating influences may be removed. Existing troubles of the drainage-apparatus should receive the proper treatment; and ectropion, if present, should be remedied. Aside from the special treatment which the particular case demands, the use of mild astringent and antiseptic solutions is indicated.

Follicular Conjunctivitis.

The **diagnosis** between this and *trachoma* is particularly important, because the granules look like those of trachoma.

Histologically, both are seen to consist of adenoid tissue; but the *clinical picture* of the diseases is very different:

Follicular conjunctivitis *occurs* in childhood and youth; trachoma is a disease of adult life.

Follicular conjunctivitis is not a serious trouble, and often terminates *without consequences*; trachoma is a serious disease, which often leaves *sequelæ* interfering with the sight and comfort of the patient. The follicles occur on the surface of the palpebral conjunctiva, and particularly in the fornix of the lower lid, and are easily brought to view on eversion of the lids.

The **cause** of the disease is uncertain; but the fact remains that follicular conjunctivitis occurs in anæmic or scrofulous individuals who often have refractive errors.

Treatment: The granules should be expressed after cocainization with the aid of a ciliary forceps. An antiseptic lotion should then be used for a few days and refractive errors corrected. Constitutional treatment is generally needed and should be prescribed.

Gonorrhœal Conjunctivitis.

General definition: This is an infectious disease due to the presence in the conjunctival sac of the *gonococcus*. When the disease occurs in adults, it receives the name blennorrhœa adultorum; when infection occurs during the parturient act or during the puerperal state, the newborn infant is said to have blennorrhœa neonatorum.

The **symptoms** are first those of an acute catarrh; but in a day or two, violent inflammation manifests itself by causing great swelling of the lids and conjunctiva. The ocular conjunctiva is swollen to the corneal edge (chemosis), so that the cornea seems sunken or depressed. Whenever during the course of a conjunctival disease, the ocular part shares in the inflammation, a microscopical examination of the secretions should be made to determine the cause. As soon as the discharge becomes free, the pain and heat subside.

The **complication** to be feared is *perforation of the cornea*, which may be due to the macerating effect of the discharge or to interference with corneal nutrition. Corneal ulcerations may heal without having produced perforation, but the vision is greatly diminished when a central opacity remains.

Incarceration of the iris in the corneal opening and panophthalmitis do occur after some perforations.

The **preventive treatment** is very satisfactory. A drop or two of a 2 per cent. silver nitrate solution in the conjunctival sac of each eye after birth of the child reduces the number of blennorrhœa neonatorum cases. In all cases precautions should be taken to prevent infection of the other eye.

The **treatment** of both forms is the same, although the disease in adults appears more severe. If seen early, when there are hyperæmia, pain, and heat in the affected parts, cold applications by iced cloths give great relief. An abortive treatment is rarely of use, and may be detrimental. A 2 per cent. solution of silver nitrate brushed over the conjunctiva two or three times daily is of value when the inflammation is not severe and discharge slight. When the swelling is considerable and the discharge excessive, stronger solutions may be employed. The cleanliness of the conjunctival sac is of the greatest importance, and to accomplish this, a bichloride solution (1 : 10,000) ; or neutralized hydrozone solution (1 : 10 or 1 : 20) ; or a saturated boric-acid solution may be employed. After the hyperæmia has subsided the use of heat instead of cold will be found of service. The periodical application of heat increases the activity of the circulatory and absorbent systems, and would hence be of great value to keep up the nutrition of the cornea, the preservation of which is the end of treatment.

The **prognosis** depends upon the physical condition of the patient and implication of the cornea.

To *relieve the pressure* on the cornea a canthotomy may be made; this also facilitates treatment.

When the *cornea is perforated*, the use of iodoform in the opening is recommended.

The *prolapsed iris* should not be snipped off. Cleanliness should be insisted on as before the perforation and atropine be employed.

The *after-treatment* of all cases consists in the use of mild astringent instillations for some weeks.

The conjunctiva after this disease is subject to other affections.

Trachoma.

General definition: This is a chronic disease of the conjunctiva, probably produced by a germ. The disease is a very serious one, for it more or less prejudices the efforts of the patient to make a living, either by interfering with vision or by necessitating prolonged treatment.

Complications: During the course of the disease, pannus, corneal ulcer, and iritis are frequent complications; and the possible *sequelæ* are distortion of the lids, symblepharon, xerosis of the conjunctiva, and corneal opacities.

The disease appears in **two forms**: The *papillary*, in which elevations are formed on the surface of the tarsal conjunctiva, more particularly of the upper lid; and the *granular*, in which granules resembling grains of boiled sago develop in the deeper layer of the conjunctiva, affecting the fornix more than the tarsal part. Both forms may occur at the same time.

Symptoms: The conjunctiva secretes a mucopurulent secretion, which causes the lids to stick together; and the irritation is such that there are usually lachrymation and photophobia. The pain may be considerable when pannus, corneal ulceration, and iritis complicate the disease. The upper lid is not fully opened because of the photophobia and thickness of the lid.

The constant irritation of the cornea by the uneven surface of the conjunctiva frequently causes the growth of a network of bloodvessels, which begins at the periphery and extends to the centre of the cornea: this is *pannus*. It produces an irregularity in the surface of the cornea; and, being superficial, can easily be differentiated from deeper corneal disease. The vessels, moreover, have a bright-red color and branch like the limbs of a tree.

Ulceration of the cornea occasionally complicates trachoma, and may occur with or without pannus.

Both corneal ulceration and pannus may cause *secondary iritis.*

Trachoma—treatment: Constitutional treatment and attention to cleanliness are of the greatest importance. The patient should be instructed to flush frequently the conjunctival sac with some weak antiseptic solution. The objects of the treatment are to reduce the conjunctival inflammation and the hypertrophy. Cleanliness and weak astringent applications, such as a 1 or 2 per cent. solution of silver nitrate, are of use when the secretion is abundant and inflammation severe. Caustics, friction, and surgical measures may be employed after subsidence of the inflammation. Cupric sulphate is usually employed three or four times weekly as a caustic, care being taken that the cornea is not touched. After eversion of the lids, iodoform gauze containing an excess of powdered iodoform or boric acid may be employed to rub the conjunctiva.

Of the *surgical methods*, expression of the granule after incision of it, is frequently employed. Expression may be accomplished with a roller-forceps or by pressing the conjunctival surface of the lids together between the thumb-nails. If the patient has a unilateral disease, he should be warned of the danger of infection to the other eye. Not only to prevent infection of the other eye, but to prevent the infection of other individuals, should the patient be careful.

Pannus—treatment: This affection frequently disappears during the treatment which is directed to the lids. In some obstinate cases, however, other measures are necessary. An infusion of powdered jequirity bean (15 to 30 grains to the ounce) should be applied in such cases to the conjunctiva three times daily for several days, exciting considerable inflammation, after subsidence of which the pannus will have disappeared. Instead of an infusion, the powdered bean in the quantity of a grain may be applied to the conjunctiva.

Ulceration of the cornea—treatment: Atropine and hot water fomentations are indicated. Copper sulphate applications to the conjunctiva and the bandage, which would retain the secretions, are *contraindicated*. *Secondary iritis* will be relieved by the application of heat and instillation of atropine.

The **sequelæ of trachoma** already mentioned demand special *treatment*. An operation can remove the irritation of trichiasis and restore the normal position of the lid. For xerosis, emollient instillations relieve the dryness due to absence of secretion. Symblepharon is not amenable to treatment. Corneal opacities may be partially removed by use of the salve of the yellow oxide of mercury.

Membranous Conjunctivitis.

Definition: This is a rare disease characterized by the formation of a membrane upon the palpebral conjunctiva of individuals whose general condition is poor.

Clinically this disease appears in **two forms**, the *croupous* and the *diphtheritic*.

Croupous conjunctivitis—symptoms: This is more common than the diphtheritic, and may be mistaken for it.

The white or grayish membrane can usually be removed without much force; systemic disturbance is slight; pain and swelling of the lids are not great; and the lids are fairly pliable. After spontaneous exfoliation of the membrane takes place, a purulent infectious discharge develops, which persists for a short time.

The **treatment** should consist in cold applications in the hyperæmic stages and the use of heat as soon as detachment of the membrane begins. A $\frac{1}{4}$ to $\frac{1}{2}$ per cent. solution of silver nitrate should be applied to the part of conjunctiva from which the membrane has been cast off. Cleanliness is to be observed in all stages of the disease.

Diphtheritic conjunctivitis—symptoms: This may be associated with faucial or nasal diphtheria, but is frequently observed alone. There is generally considerable constitutional disturbance, and the picture of the disease is that of an aggravated form of croupous conjunctivitis. The redness and swelling of the lids are extreme, and the pain is great on manipulation of the lids, which become as hard as a board, rendering eversion an impossibility. The ocular conjunctiva may become hyperæmic and corneal complications are likely to occur.

In the **treatment** sustaining measures should be prescribed. When the presence of the Klebs-Loeffler bacillus can be demonstrated, antitoxin suggests itself. Attention to the cleanliness of the conjunctival sac, so far as it does not necessitate rude handling of the board-like lids, should be enjoined. Hydrozone of the strength tolerated by the patient should be used for its antiseptic effect. The use of heat should be faithfully continued in order to increase the nutrition of the cornea and prevent complications which indicate the use of atropine. When opposing surfaces of the mucous membrane are denuded, the lids should be frequently moved over the surface of the ball to prevent symblepharon. Vaseline may be employed for the same purpose.

Phlyctenular Conjunctivitis.

Definition: This is a manifestation of scrofula, anæmia, or faulty assimilation, especially frequent in *children*.

The **lesion** is a *vesicle*, which is seen upon the ocular conjunctiva or under the epithelial layer of the cornea. In some cases it seems to be associated with nervous derangement.

The disease varies in severity of the **symptoms**, owing to different susceptibilities of the nervous system and the existence or not of pressure by the exudate.

In some cases the only manifestation is a slight redness of the conjunctiva about the vesicle, producing no other symptoms.

On the other hand, lachrymation, photophobia, blepharospasm, ectropion, eczema of the cheek, excoriation and ulceration of the lower lid, and corneal ulcers may be present.

Many patients, if left alone, bury their heads in pillows or seek dark corners to avoid the light. The appetite is usually poor or capricious.

The **treatment** depends upon the severity of the symptoms. Constitutional treatment is of prime importance, hæmatics being indicated. The syrup of iodide of iron is particularly useful. In some cases the salicylates are efficacious. When the symptoms are *severe*, a thorough examination, even if it require the use of an anaesthetic, should be made in order to determine the presence of corneal ulcers, which, if neglected, might end disastrously. *Blepharospasm* yields to immersion of the little patient's head in cold water. The use of lid-retractors or a spring speculum for several minutes a couple of times daily is also of value. *Excoriations* yield to cleansing of the conjunctival sac and the application of zinc salve. *Fissures* of the outer canthus heal promptly after use of the silver nitrate in strong solution or in stick form. *Ulceration of the cornea* is best treated by cleansing the infected surface and cauterization with the galvanic cautery or carbolic acid. The preliminary instillation of cocaine is sufficient for the cauterization when the patient is not intractable.

The antiseptic effect of calomel dusted in small quantity in the eyes; or of a salve of the yellow oxide of mercury (1 grain to the drachm) rubbed about the conjunctival sac, is of value. Atropine should be employed when there are corneal complications.

When the disease is very obstinate, change of climate and correction of existing refractive errors are of value. In some cases attention to the nasal mucous membranes effects a speedy cure. When the corneal ulcers are superficial, eserine might be tried when this complication does not yield after use of atropine.

Vernal, or Spring, Catarrh.

Symptoms: This is a rare disease appearing with the first warm days of spring and lasting until fall, characterized by circumcorneal hypertrophy of the bulbar conjunctiva, and granular elevations of the tarsal portion. The conjunctiva appears uneven and thickened and has a milky appearance.

The **treatment**, which unfortunately is only palliative, consists in mild astringent instillations and antiseptic lotions.

Specific Ulceration of the Conjunctiva.

This may be of *tuberculous* or *syphilitic* origin.

The **tuberculous ulcer** may be a primary affection. It has ragged edges, and may from a small beginning involve the entire conjunctiva and cornea. In consequence of extensive ulceration symblepharon may result. Pain is not present unless the cornea be involved. *Treatment* should be both general and local. Cleansing antiseptic solutions should be frequently employed. Small ulcers may be cauterized, but larger ones demand milder treatment.

Syphilitic ulcers may represent the primary lesion, in which case the induration and glandular involvement make the diagnosis easy. When the ulcer is soft recognition is not so easy. Caustics should not be used, but the *antisyphilitic treatment* instituted.

Xerosis of the Conjunctiva.

Symptoms: This disease is characterized by dryness and hardness of the mucous membrane whereby the natural secretions are materially diminished or abolished. It may be the result of cicatricial changes incident to trachoma, traumatism, or diphtheritic conjunctivitis; and of exposure of the conjunctiva, such as occurs in ectropion and lagophthalmos. It may also be *primary*, particularly in children whose health is of the worst. In these cases, besides the dryness and accumulation of mucus, night-blindness and corneal complications occur.

Treatment: When the general health is at fault, tonics and constitutional treatment may improve the disease. Infants usually die in spite of all treatment. When due to cicatricial changes, the use of emollients is palliative. Cure there is none. When caused by ectropion or lagophthalmos, operative interference is of value.

Pterygium.

General definition: Pterygium is a triangular-shaped, vascular thickening of the bulbar conjunctiva, which finally invades the cornea, occurring usually over the situation of the internal rectus. The apex or head of the pterygium is near or upon the cornea, the base toward either the inner or the outer canthus, according to the side from which it starts. A pterygium may for a long time remain stationary or grow until its apex covers the pupil, interfering seriously with vision. A stationary pterygium is thin, pale (because it is not very vascular), and tendinous in appearance. When *progressive* it is thick and the edges alone are whitish and non-vascular.

The **etiology** is obscure, but irritation seems to be a factor.

Treatment: A progressive pterygium should be removed without delay. A stationary one when there are no symptoms may be left alone. For removal of the pterygium the ligature, transplantation, or excision may be employed. When very large, so that after excision it is impossible to coapt the edges of the conjunctiva, the use of skin-grafts is of value.

Subconjunctival Hemorrhage.

Etiology: When involving the bulbar conjunctiva particularly it occurs after operations such as tenotomies; and is sometimes a manifestation of conjunctival disease. During the course of whooping-cough it is not uncommon. Sneezing and vomiting as well as coughing may cause it. In old people, in whom the walls of the bloodvessels have degenerated, rupture may occur, producing subconjunctival hemorrhage. Fracture at the base of the cranium sometimes permits the blood to find its way into the orbit, thus showing itself under the conjunctiva.

Treatment is given more to satisfy the patient than to hasten the absorption. Any placebo will answer the purpose.

Chemosis.

Chemosis, or *œdema of the conjunctiva*, the ocular part particularly, is usually symptomatic. In severe inflammations of the lids or orbital tissues, glaucoma, conjunctivitis, iritis, or iridocyclitis, it may be a prominent symptom. Idiopathic chemosis does, however, occur, probably resulting from a disturbance of the circulation. Chemosis at times causes an elevation of the conjunctiva around the cornea, which appears sunken. The color of the thickened conjunctiva is not like that of the dark-red extravasation observed in subconjunctival ecchymosis, but is of a pale-pink or yellow.

Treatment: To relieve the œdema, punctures or incisions may be called for when the nutrition of the cornea is interfered with. Applications of hot compresses facilitate the absorption of the fluid.

THE SCLERA.

Anatomy.

The sclera and cornea form the **outer coat** of the bulb, the sclera representing the posterior five-sixths.

The optic nerve enters the posterior part through a perforation known as the **foramen scleræ.**

The **external scleral surface,** which is smooth, with the exception of the insertions of the recti and oblique muscles, is connected to the *capsule of Tenon* and the *ocular conjunctiva.*

The **internal scleral surface** is loosely connected to the *choroid* and *ciliary body* by the *lamina fusca.*

The **outer layers** of the sclera reinforce the outer layers of the optic nerve-sheaths.

The **middle layers** of the sclera cross the entrance of the optic nerve, forming the *lamina cribrosa*, the numerous perforations of which transmit the optic-nerve fibres.

Through the large central opening, the **porus opticus,** passes the *arteria centralis retinæ.*

The cornea and sclera are similar in structure, the only difference being an **opacity** of the fibrous connective tissue of the *sclera.*

The sclera receives its **blood-supply** from the long, short, and anterior ciliary arteries. The anterior ciliary arteries pierce the sclera a short distance behind the corneal margin. These also supply the iris and ciliary body, and form largely the *anterior ciliary zone.*

Inflammation of the iris, ciliary body, and choroid produces engorgement of this zone, and hence the **anterior ciliary zone is** then easily seen just back of the sclerocorneal junction.

Congestion of the anterior ciliary zone is easily differentiated from that of the ocular conjunctiva by producing pressure of the lid on the bulb, when the conjunctival congestion temporarily subsides, while the other remains unchanged.

Schlemm's canal, or *circulus venosus*, is a sinus situated at the sclerocorneal junction near the internal surface of the sclera. It is in communication with the anterior chamber, and empties its contents into the anterior ciliary and scleral veins. The drainage of the eye is effected through this canal, and hence *glaucoma* can be caused by conditions which interfere with the function of this canal.

Congenital pigmentation of the sclera is not uncommon and might be mistaken for pigmented patches of the conjunctiva.

Neoplasms of the sclera are very rarely primary; but secondary involvement frequently occurs. Fibromas, sarcomas, and osteomas primarily occurring in the sclera have, however, been observed.

Injuries.

Contusions of the sclera may be of serious import to the eye, since intraocular complications frequently coexist. When the contusion is very violent and the elasticity of the eyeball insufficient, rupture of the globe results. The lens and iris may be expelled when the conjunctiva is torn. Rupture usually occurs at the inner and upper margin of the cornea, and the eye is generally lost, although attempts to save the eye may at first seem feasible. Panophthalmitis frequently occurs, and at times it is necessary to enucleate the injured eye when pain and irritation suggest a possibility of sympathetic inflammation.

Burns may affect the sclera.

Wounds of the sclera vary in kind and severity, but are very serious when a puncture is made by any unclean agent.

When the *ciliary body* is injured in consequence of a wound of the sclerocorneal margin, sympathetic inflammation and iridocyclitis are likely to occur. The danger of the injury is increased in all cases when the *retina*, *choroid*, or *vitreous* is involved.

Foreign bodies seldom lodge in the sclera: they either rebound from the eye or pass through the globe. Foreign bodies in the eye are always a menace, and should, if possible, be removed. Particles of iron or steel may occasionally be removed by the use of an electromagnet, and the attempt should be made, if there is any possibility of saving the eye. Very often it is an act of charity to enucleate an eye containing a foreign body, since the patient is thereby spared great suffering and the possible loss of the uninjured eye from sympathetic processes.

DISEASES OF THE SCLERA.

Episcleritis.

Symptoms: This is a chronic disease with a tendency to recurrence affecting the external layer of the sclera in such a way that a sharply limited elevation near the limbus develops. This is not movable, and pressure over the surface does not cause the complete emptying of the bloodvessels seen in conjunctival congestion. The portion of the conjunctiva over the affected area is somewhat congested, but there is little or no discharge. Lachrymation, photophobia, and pain sometimes of a severe type are the prominent symptoms.

Diagnosis: It may be mistaken for phlyctenular conjunctivitis at the beginning; but the fact that it is a disease of adults more particularly, whereas children are more often subject to phlyctenular troubles, should be kept in mind.

The **cause** of episcleritis probably lies in the tendency to gout or rheumatism or the existence of syphilis, and when this has been determined the proper treatment should be instituted.

Local treatment consists of scarification at its incipiency, and the use of dry heat and atropine. Subconjunctival injection of a normal salt or bichloride solution (1 : 1000) is of use in some cases.

Scleritis.

Scleritis is a chronic disease in which the deeper layer of the sclera is involved, in consequence of which the ciliary body, iris, choroid, and cornea are likely to become secondarily affected.

A thickening of the sclera takes place at the site of the inflammation, and after the disease has run a tedious course, absorption of the exudate takes place, leaving the sclera much thinned, so that when the intraocular tension is increased a bulging, or **staphyloma**, as it is termed, results.

The **causes** of scleritis are uncertain, yet manifestations of syphilis, rheumatism, gout, or tuberculosis are frequently evident. Females are more frequently affected than males, and menstrual difficulties may have some causal influence.

Complications: Scleritis may cause opacities of the cornea, seclusio pupillæ (complete circular adhesion of the iris to the lens), opacity of the lens or vitreous, myopia, and secondary glaucoma.

The **treatment** consists in attention to the general health and local application of hot cloths. Rest is absolutely necessary in some cases, and the use of atropine is indicated in ciliary and iritic complications. An iridectomy is indicated when there is no communication between the anterior and posterior chambers, in order to prevent secondary glaucoma. The operation should not be undertaken until inflammatory manifestations have subsided.

Ectasia.

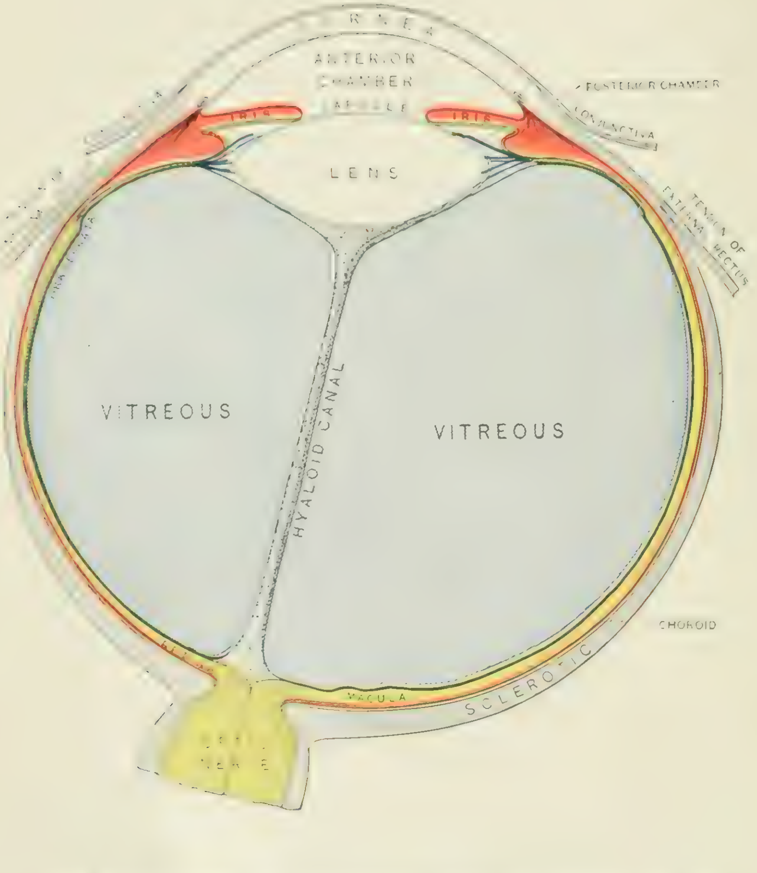
Partial ectasia of the sclera is a bulging due to thinning, increase of intraocular pressure, or the combination of these conditions, and may occur at the anterior part of the globe, the equator, or the posterior part.

Complete ectasia involves the whole eyeball, and is either *congenital* or occurs in *infancy*.

When *congenital*, the filtration angle is either absent or reduced in size because of adhesion of the iris and the cornea; and the sclera, being elastic in infancy, yields to the increased pressure induced by the interference with the drainage through Schlemm's canal.

An **anterior ectasia** or **staphyloma** involves either the sclera covering the ciliary body or that immediately in front of it. The *cornea* is always more or less displaced.

PLATE II.



The Right Eye in Horizontal Section, showing the upper surface of the lower segment. Diagrammatic. (Testut.)

Posterior staphyloma may be due to posterior sclerochoroiditis. When congenital it causes a malignant form of myopia. Myopia of high degree—more than 10 D.—is also associated with a posterior staphyloma.

The **treatment** of *anterior* or equatorial *ectasias* is an iridectomy. Pain and cosmetic reasons demand an enucleation when other means of help fail.

Posterior staphylomas are not amenable to treatment, but the coexisting myopia should be corrected.

Complete ectasia, which is known as *hydrophthalmos* or *buphthalmos,* is sometimes progressive, so that the eyeball assumes enormous dimensions, and the sight is lost.

Treatment is of no avail in such a case, and when the condition is stationary interference is not necessary.

THE CORNEA.

Anatomy.

The cornea and sclerotic represent the **fibrous envelope** of the eye (Plate II.). The cornea forms the anterior transparent one-sixth, the sclerotic the posterior five-sixths of the eyeball. There is no abrupt line of demarcation between them. The cornea lies in the sclera, which externally slightly overlaps its margin. The cornea is ellipsoidal in shape, the horizontal diameter being slightly longer than vertical.

The cornea is divided into **five layers,** from without inward, as follows: 1, epithelial layers; 2, anterior limiting membrane, or membrane of Bowman; 3, true corneal substance; 4, the posterior limiting membrane, or elastic lamina; and 5, the endothelial layer.

The **epithelial layer** consists of three kinds of epithelial cells, squamous externally, then polyhedral, and columnar internally.

The **membrane of Bowman** is a fibrillate dense layer which is like the corneal layer proper, except that it contains no corneal corpuscles.

The **true corneal substance** constitutes the larger part of the cornea and is one-twenty-fifth of an inch in thickness. It consists of a *ground-substance,* made up of lamellæ which cross each other at different angles, united by an interfibrillar *cement-substance*; and of *corneal corpuscles*, which occupy the space between the lamellæ.

The *interlamellar spaces* are connected with one another by narrow canals.

The **posterior limiting membrane**, or Descemet's, is a thin membrane intimately connected to the posterior surface of the true corneal substance. It is the toughest of the corneal layers, and is quite brittle. It is continuous with the pectinate ligament of the iris at its margin.

The **endothelial layer** is a single stratum of polygonal flattened cells which lines the internal surface of the posterior limiting membrane.

The long and short **ciliary nerves** divide into about forty branches, which pierce the sclera to enter the cornea at its periphery.

The cornea has no bloodvessels, but is exceedingly rich in nerves, which do not interfere with its transparency, because they have no sheaths. The nerves form two networks: the one supplying the membrane of Bowman and the external epithelial layer; the other the posterior layers of the cornea. The anterior denser network is situated just behind Bowman's membrane, the posterior delicate network is just in front of Descemet's membrane.

The cornea gets its **blood-supply** from episcleral branches of the anterior ciliary arteries and from the lachrymal and infratrochlear arteries which supply the conjunctiva. These divide into a capillary network in the true corneal substance at the periphery of the cornea, and the nutrient material passes through the canalicular lymph-system to all parts of the cornea.

The cornea has a greater **refractive power** than any other part of the dioptric system of the eye, and hence claims great attention in correction of refractive errors. The *index of refraction* of the cornea is 1.3523.

Congenital Abnormalities of the Cornea.

Macrocornea is a condition in which the cornea is enlarged, occurring in buphthalmos.

Microcornea is a small cornea, and is usually seen in microphthalmic eyes.

Opacities of the cornea may be congenital and vary in size and intensity. Complete opacities are more often associated with buphthalmos, although they do sometimes occur in microphthalmic eyes. Partial opacities show great variations of size and degree.

Conical cornea, or *keratoconus*, may be congenital, although its development sometimes extends over many years. The cornea protrudes and is conical in shape, the apex being usually the thinnest part and subject to opacity. The increased length of its axes renders the eye myopic, and the irregular curvature of the cornea produces astigmatism.

Examined with the *ophthalmoscope*, one observes a central red reflex which is surrounded by a circular shadow. *Treatment* is of little use.

Dermoid growths of the cornea are invariably congenital and involve more or less of the ocular conjunctiva. They are usually associated with a coloboma of the lid or other anomaly.

When they irritate the eye, their removal is necessary. This should be thoroughly done and the conjunctiva brought over the denuded conjunctival surface. Where too much conjunctiva has been removed, skin-grafts may be employed as in treating very large pterygia.

Neoplasms rarely occur primarily in the cornea. Carcinomas and sarcomas starting in the limbus of the conjunctiva invade the cornea secondarily.

Injuries.

Foreign bodies of endless variety may lodge upon or in the cornea. After cocainizing the eye these should be removed. When *superficial*, the spud is the best instrument ; when *deep*, the needle is best suited to the case.

When there is danger of pushing the foreign body into the anterior chamber, the lids should be kept open by a speculum, the eyeball held by a fixation forceps, and a Beer cataract-knife pushed through the cornea behind the foreign body. With this as a guard, attempts at removal may be safely made.

A mild antiseptic wash is useful in most cases after removal of the foreign body, and occasionally, when there is considerable reaction, atropine may be necessary to rest the eye.

Injuries of the cornea of whatever kind are serious according to their depth and location. When only the epithelial layer is involved no scar results, which is the case when the substantia propria is injured. A central injury producing a central opacity impairs the sight according to its intensity.

Contusion of the cornea results in a loss of epithelium, which after a few days' rest is regenerated.

Wounds: *Penetrating wounds* of the cornea are serious according to the location and complications. Wounds at the sclerocorneal junction, from possible injury to the ciliary body, leading to sympathetic trouble and prolapse of the iris, are very dangerous.

Traumatic cataract, dislocation of the lens, prolapse of the iris or vitreous, or a coincident injury of the choroid and retina may complicate a corneal wound.

When infected the danger of corneal wounds is increased, so that the outcome may be a panophthalmitis.

Treatment of injuries: In simple wounds cleanliness is all that is necessary. When there is danger of iritis, atropine should be used. A prolapsed iris should be snipped off.

Foreign bodies penetrating the eyeball more frequently pass through the cornea than the sclera. The wound can be seen by oblique illumination. The object may remain in the anterior chamber, become imbedded in the lens with or without wounding the iris, pass through the lens or zonula, and either remain in the vitreous chamber or penetrate the coats of the eye. The danger depends upon the presence of infection and damage done to the structures. A foreign body should in every case be removed.

Burns and **corrosion** of the cornea frequently occur. When superficial they cause no impairment of sight; when deep, the cornea may become opaque. The *treatment* consists in cleanliness of the conjunctival sac, the instillation of atropine, and rest.

DISEASES OF THE CORNEA.

General Diagnosis.

Diseases of the cornea are of the greatest importance, since the acuity of vision depends upon the corneal transparency.

This is more or less impaired in *corneal diseases*, which constitute one-fourth of eye-diseases. An early recognition, therefore, of corneal diseases, as well as those of the conjunctiva which implicate the cornea, is imperative in order that by proper treatment the damage to vision be reduced to a minimum.

In all forms of **keratitis** the cornea should be carefully examined with a view of determining the presence of scars, opacities, irregularities of the surface, and the formation of superficial or deep-seated vessels.

Dense opacities are easily recognized, but often lateral illumination is necessary to demonstrate the existence of others.

When there is **loss of substance** involving the corneal surface, an irregularity results which can be recognized by noting the reflection of the window before which the patient should be seated. When the cornea is normal, the picture of the window will be perfect in all positions of the eye ; if any irregularity exists, the reflected image of the window will be broken or imperfect.

Superficial vessels occur in **pannus**; deep vessels in **parenchymatous keratitis**; accordingly the diagnosis of superficial and deep forms of keratitis is facilitated by noting the situation of the vessels.

Complications of corneal disease: Photophobia, blepharospasm, lachrymation, or diminished visual acuity, intraocular tension, and sensibility of the cornea may be observed.

Corneal Ulcer.

Etiology: A corneal ulcer is the result of an infiltrate which, becoming infected, causes an exfoliation of the superficial epithelium over the site of the inflammation, and a destruction of more or less of the corneal tissue according to the size and depth of the invaded part.

When the ulcer shows evidence of loss of tissue within an uneven opaque infiltrate, it is in the **progressive stage,** which is characterized by pain, lachrymation, photophobia, and ciliary congestion.

When the formation of the infiltrate stops and the infected tissue is cast off, the ulcer is in the **regressive stage,** in which the signs of irritation diminish or cease in proportion to the completeness of the exfoliation. Bloodvessels from the limbus of the cornea develop to the edge of the ulcer and the regeneration of the tissue begins. This scar-tissue, because of its irregularity of arrangement and compactness, is not transparent, and constitutes the *opacity.*

When the destructive process continues through the cornea, a **perforation** results and the aqueous trickles out, the intraocular tension being reduced and the corneal nutrition increased.

The **consequences** of a perforation are sometimes very serious, since prolapse of the iris, anterior synechia, fistula, anterior polar cataract, dislocation of the lens, intraocular hemorrhage, and infection, resulting in an iridocyclitis or panophthalmitis, may occur.

Prognosis: Opacities through the entire thickness of the cornea remain forever. The patient's age and depth of the ulcer modify the prognosis of corneal opacities. The younger the individual, and the less the corneal tissue involved, the better is the prognosis.

A corneal ulcer is said to be **primary** when it occurs in consequence of some corneal disease; and **secondary** when it is the result of some conjunctival disease.

The **treatment** of corneal ulcers depends upon the cause; but since there is always infection at the bottom of every ulcer, cleanliness is of importance.

Diseases of the conjunctiva, as trachoma and blennorrhœa neonatorum or adultorum, should receive their proper treatment when the ulcer is secondary to these affections. Should *perforation* of the cornea occur, treatment should be continued, with extra care not to injure the cornea by contact with any caustic employed. Copper and lead should not be used in the treatment of conjunctival disease when the cornea is secondarily affected. The bandage is useful in the treatment of corneal ulcers except when the secretions are considerable. Atropine is also of great service. When the ulcer appears rapidly progressive, the application of moist heat, antiseptics, the cautery (electro- or actual), pure carbolic acid, or paracentesis must be resorted to.

The serious results of a *prolapsed iris* complicating a perforated cornea should be managed according to the size of the perforation and the duration of the prolapse.

In a favorable case of small perforation, there is not any adhesion of the iris to the cornea (anterior synechia).

In other cases a few shreds of the iris remain forever adherent.

A large perforation allowing prolapse of the iris is best treated in a recent case by excision of the prolapsed iris in such a way that none of it remains in the corneal opening. This is accomplished by releasing the iris from the edges of the cornea, and after pulling the prolapsed iris well out of the perforation, by cutting it off.

A *corneal fistula* is treated by rest of the patient on the back, employment of eserine; and in case of failure, by freshening the corneal edges with a knife or galvano-cautery.

When a corneal ulcer is in the stage of *regeneration*, the use of the yellow oxide of mercury salve (gr. viij to the ounce) in a measure reduces the opacity.

Corneal Abscess.

Etiology and pathology: A *corneal abscess* forms in the substance of the cornea, usually in the central part. At the beginning it is surrounded by healthy corneal tissue, of which the overlying portion is slightly depressed. Its margin is the more opaque part. An abscess is due to an abrasion of the epithelial layer of the cornea and the infection of the wound by microörganisms, which may be present in the secretions or in foreign bodies. When the pus gravitates between the corneal layers to the lower corneal margin it constitutes an *onyx*. When the abscess communicates with the anterior chamber a *hypopyon*, or accumulation of pus in the anterior chamber, results. When the overlying corneal tissue breaks down an *ulcer* is formed. An abscess is a very serious disease, since its most favorable termination is a central opacity which is permanent.

In other cases, posterior synechiæ, obliteration of the pupil, or panophthalmitis may result.

The **symptoms** of corneal abscess are generally severe: photophobia, ciliary injection, œdema of the lids, and pain. An *iritis* always complicates an abscess and posterior synechiæ often occur.

The **treatment** of corneal abscess depends upon the cause and severity. In simple cases, atropine, the bandage, and moist heat suffice. In all cases the disease producing the infective secretion should receive attention. In severe cases cauterization with pure carbolic acid or the electrocautery is useful; and a paracentesis frequently repeated relieves tension and has a favorable influence. After inflammatory signs have subsided, an *iridectomy* is often indicated to make an artificial pupil.

Keratitis—Special Varieties.

Keratitis e lagophthalmo is due to want of proper protection and lubrication of the cornea. The insufficient secretions evaporate, leaving the corneal epithelium dry, and after exfoliation, infection of the cornea, producing an ulcer or abscess, results. The termination of such a keratitis is that of ulcer or abscess.

The *treatment* of the condition producing the lagophthalmos is of primary importance. In light cases, a temporary bandage should be used to protect the eyes; in others a tarsorrhaphy is necessary.

Keratitis neuroparalytica is due to paralysis of the trifacial nerve. In spite of *treatment*, which consists in the use of atropine, bandage, and moist heat, in consequence of the corneal opacity the sight is lost.

Keratomalacia is a disease of the cornea affecting children, and because of faulty nutrition of the patient the cornea undergoes degenerative changes resulting, if the life is not lost, in opacities of the cornea.

The *treatment* consists in measures to improve the general condition, and the local use of moist heat and the bandage.

Pannus may be a complication of trachoma or phlyctenular conjunctivitis. When due to trachoma, the upper part of the cornea is the usual site, possibly because of irritation of the winking upper lid; but when it accompanies phlyctenular disease, any part of the cornea may become involved.

In the course of *treatment* for phlyctenular conjunctivitis, the pannus, when present, almost entirely disappears, differing in this respect from the pannus of trachoma.

Vesicles and **blebs** occur on the cornea, and damage to the cornea depends upon the cause. Vesicles which accompany febrile diseases leave no bad consequences, as a rule, when proper attention to cleanliness has been observed. Should an ulcer occur, atropine, moist heat, and the bandage are indicated.

Vesicles are sometimes seen on the cornea, complicating herpes zoster ophthalmicus. The *treatment* is the same as above, but the consequences are likely to be more serious, since ulceration more frequently occurs.

Vesicles and bullæ sometimes develop in the cornea of eyes useless from large opacities, iridocyclitis, or glaucoma. The interference with the lymphatic circulation produces œdema of the cornea, and vesicles or blebs, according to the size of the elevation, occur. The *treatment* is not very satisfactory, since frequent recurrence of the condition is the rule. When the eye is sightless, an *enucleation* is often the best treatment.

Parenchymatous Keratitis.

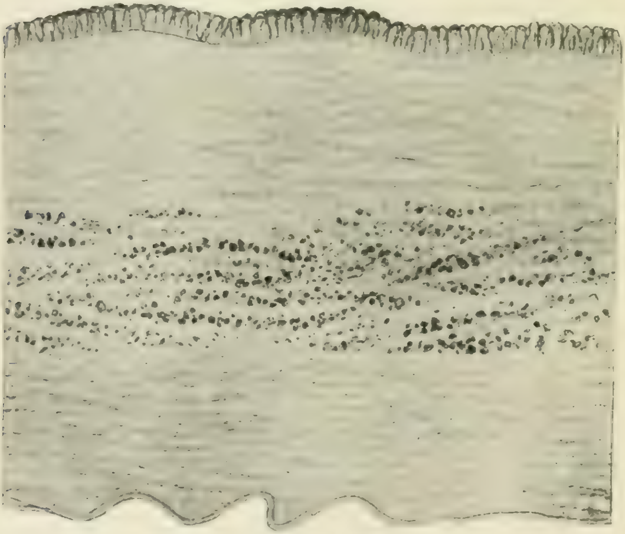
Parenchymatous or **interstitial keratitis** (Fig. 15) is a chronic inflammation of the deeper layers of the cornea, usually due to hereditary syphilis, occurring generally between the ages of six and twenty.

The **etiology** is sometimes obscure; may be scrofula, but is in most cases hereditary syphilis. Acquired syphilis very seldom causes primary disease of the cornea.

In many cases evidences of syphilis are striking, one or more of the following being present: a protruding frontal bone, concave nose, poorly developed upper jaw, scars at the angles of the mouth, incisor teeth with concave biting edges, enlarged lymphatic glands, impairment of hearing, and thickening of the periosteum of the long bones, particularly the tibia.

The **clinical picture** of the disease varies with the intensity and the place of origin, whether it be at the margin or at the centre of the cornea. The disease usually extends from the *margin* to the centre of the cornea, in which case the opacity, which is in the deeper layers of the cornea, gradually progresses until the entire cornea presents a milky appearance. The network of bloodvessels which supply the cornea send out branches into the substantia propria. These vessels may take in a large part of the cornea and give it a dull reddish hue. They can be differentiated from the bloodvessels of pannus by the fact that they stop suddenly at the limbus, they have a brush-like appearance, are not distinct, and the surface of the cornea is even, but without lustre.

Fig. 15.



Section of interstitial keratitis. (Wedl.)

Parenchymatous keratitis beginning at the *centre* of the cornea is usually more severe. Several whitish opaque spots are seen in the deeper layers of the cornea, and these finally coalesce. The vision is necessarily very much impaired, owing to the central location of the opacity, and the pain, congestion, and photophobia are extreme. It will be found difficult at times to dilate the pupil with an atropine solution, but when successful the torn vessels of the iris cause some hemorrhage into the anterior chamber. In all cases, however slight, there is some involvement of the uveal tract, from hyperæmia of the iris to a plastic iridocyclitis, which may terminate in atrophy of the bulb.

Treatment: During the inflammatory stage of keratitis parenchymatosa, atropine, moist heat, and protection from light are indicated. After subsidence of the inflammation, the use of some stimulating salve, as the yellow oxide of mercury, is of use to dissipate the corneal opacity. The constitutional treatment is also very important, and syphilis, which is usually the cause of the affection, should be treated with mercurials and iodides. The nourishment of the patient should receive attention, and with this end in view the food should be simple and nutritious. Milk and eggs are of great value in these cases ; and when the excretions are normal large amounts of milk are very potent. At times enormous doses of the iodides are not only tolerated, but their favorable influence is manifested by rapid amelioration of the symptoms.

Keratitis parenchymatosa circumscripta is an inflammation involving the middle and deeper layers of the cornea. It differs from the parenchymatous keratitis in that it is circumscribed, the formation of new vessels is slight if there be any, the uveal tract is but slightly involved if at all, and the disease affects only adults.

The **etiology** is generally obscure, but exposure to cold, malaria, or traumatism has a causal effect in some cases.

The **local treatment** is that of any keratitis—atropine, bandages, and moist heat. The *general treatment* is the use of salicylates or quinine when cold or malaria seems to be the cause.

A **keratitis** may be the result of any condition which interferes with the integrity of the endothelial layer of the cornea. The aqueous humor may, by infiltration, cause a dulness of the cornea. Exudates in the anterior chamber, prolapse, cyst or tumor of the iris, and a dislocation of the lens into the anterior chamber are conditions which may lead to keratitis from absorption of the endothelium.

Defects of the Cornea.

Opacities of the cornea may be due to corneal inflammations, old age, or intraocular diseases.

Opacities due to suppurative or non-suppurative corneal disease are classified according to their density as *maculæ* when faint and *leucomæ* when dense.

When the surface of the cornea is depressed over a small scar from insufficient tissue-regenerations, a so-called *facette* results.

When a large part of the corneal surface is flattened in consequence of a large scar, the condition is known as *aplanatic cornea.*

In old age, an opaque semicircle develops near the sclero-corneal margin of the upper half of the cornea, and is soon followed by a similar line in the lower half. These often unite, forming a complete circle, and the segment is known as *arcus senilis* or *gerontoxon.*

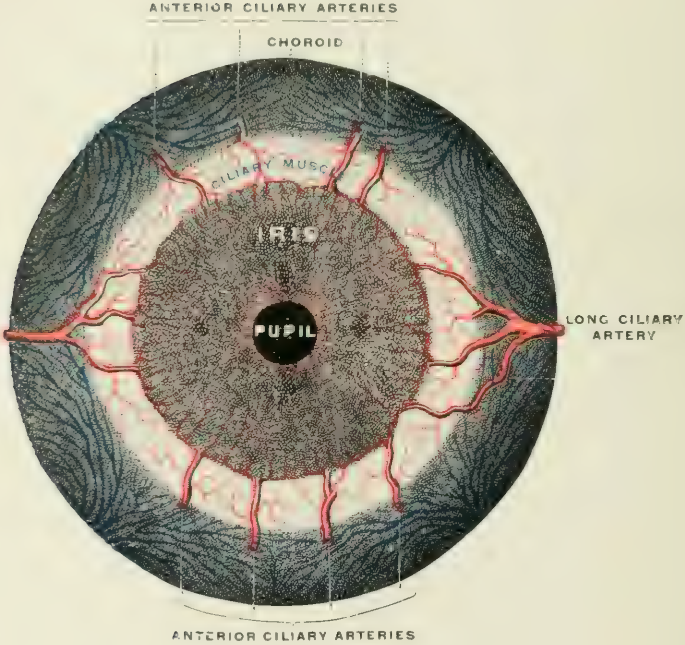
In consequence of glaucoma or an iridocyclitis, corneal opacities may also occur; but these are not very significant, since they occur in sightless eyes.

The *treatment* of opacities consists in the use of irritants and the stereopaic spectacles, the correction of the refractive errors, and an iridectomy to establish an artificial pupil, as the case may demand. Opacities which by their whiteness are disfiguring may be tattooed with India ink. Only old firm scars occurring in eyes not attacked by iridocyclitis should be tattooed.

An **ectasia** (or bulging) of the cornea may be the result of some inflammatory corneal disease; or may occur without any antecedent corneal affection.

Staphyloma and **keratektasia** are usually the result of ulcerative keratitis. A *staphyloma* is due to a perforation of the cornea in which the iris becomes incarcerated, forming a part of the scar. A *keratektasia* is due to an ulceration which never perforates the cornea, but causes material thinning of it

PLATE III.



Iris, front view. (Testut.)

at the site of the bulging. The corneal surface over the keratektasia is always opaque, and thus differs from keratoconus and keratoglobus, both of which present ectasias without antecedent disease.

The *treatment* of staphyloma, if partial, aims to prevent its progress, and this can at times be effected by paracentesis frequently repeated and a pressure-bandage; or an iridectomy with firm pressure. When complete, an incision through the protruding cornea should be made and a part of the edges excised, in order that the cicatrix may become more flattened. Enucleation is at times necessary to relieve pain and prevent sympathetic inflammation.

Keratektasia is not amenable to treatment.

Keratoconus is a rare disease, the cause of which is not understood. It is characterized by a thinning of the central part of the cornea and a conical projection.

Treatment: Tonics and myotics have had some influence in checking the disease. Cauterization or excision of the apex and an artificial pupil by performing an iridectomy have been successful in a few cases.

Keratoglobus is a feature of hydrophthalmos, in which the cornea is larger and more bulging than normal.

THE IRIS.

Anatomy.

The choroid, ciliary body, and iris form the **uveal tract.**

The ciliary body, or middle zone, connects the choroid, or posterior zone, and the iris, or anterior zone.

The iris **arises** from the anterior surface of the ciliary body (Plate III.), being reinforced by some fibres of the ligamentum pectinatum iridis and the inner wall of Schlemm's canal.

The **pupil** is the central perforation of the iris, and is subject to variations of size. The iris divides the aqueous chamber, the space in front of the lens, and suspensory ligament into an anterior and a posterior chamber, between which there is normally free communication through the pupil.

The **anterior surface** of the iris is divided by an uneven circular line into two zones, a pupillary and a ciliary. Near the circular line in the ciliary zone are numerous *facets;* nearer the periphery are several *rings* or furrows concentrically arranged.

The *lesser arterial circle* of the iris produces the uneven circular line, and the *radiating lines* extending from the periphery to the pupil represent small vessels in the vascular layer.

The **iris consists** of an anterior endothelial layer, stroma, or vascular layer, a muscular layer, and a pigment-layer.

The **anterior endothelial layer** is a continuation of a part of Descemet's membrane, and is wanting over the facets of the ciliary zone.

The **stroma, or vascular layer**, contains the arteries and veins, between which are round and branched cells held in place by connective-tissue fibres.

The two *long posterior ciliary* arteries supply the iris, forming two arterial circles; the peripheral one being the circulus arteriosus iridis major, the radiating branches of which near the pupil form the second circle, circulus arteriosus iridis minor.

The amount of **pigment** in the cells of the stroma determines the color of the iris, except in the case of albinism, when there is no pigment present anywhere in the iris. The iris appears blue when there is no pigment in the stroma, gray when there is a slight amount, and brown when there is very much.

The **muscular layer** contains two kinds of fibres, one constituting the *sphincter pupillæ*, the other the *dilator pupillæ.* These fibres lie in the posterior part of the vascular layer.

The *sphincter pupillæ* is supplied by the motor oculi, of which direct or reflex stimulation produces contraction of the pupil. Paralysis of the third nerve causes dilatation of the pupil.

A branch of the cervical sympathetic to the lenticular ganglion supplies the *dilator pupillæ.* Stimulation of this nerve causes dilatation, paralysis, contraction of the pupil.

Not only the sphincter of the iris, but the ciliary muscle and the internal recti are supplied by the third nerve, and since the centres of accommodation, pupillary reaction, and convergence are intimately connected in the floor of the fourth ventricle, normal convergence and accommodation are accompanied by a contracted pupil.

This phenomenon is known as the *associated reaction* of the pupil.

Moreover, when light stimulates the retina of one eye (the other being covered), causing a contraction of the pupil, a similar contraction of the pupil of the covered eye also occurs. This is known as *consensual reaction*, and is due to the fact that the stimulus of one eye, owing to the decussation of the optic nerve fibres in the chiasm, passes along both optic tracts to the nuclear centres in the fourth ventricle and back again by way of the third nerve to both irides.

The *Argyll-Robertson* pupil is one which does not react to light, whereas convergence and accommodation cause reaction, and is due to a change in the fibres of the optic tract which go to the fourth ventricle.

Congenital Abnormalities of the Iris.

The posterior epithelial layer and the cells of the vascular layer, or stroma, contain the *pigment* which determines the color of the iris. When there is no pigment in the posterior epithelial layer it is always absent in the stroma.

This condition occurs in **albinism.** The iris of albinos does not sufficiently exclude an excess of luminous rays, and hence these people half close their lids in bright light. The vision is poor, and nystagmus frequently complicates the trouble.

Corectopia is the condition in which the pupil in one or both eyes is more or less eccentric. Slight alterations of position frequently occur; extreme cases are rare, and there are usually complications of dislocated lenses.

Microcoria is a small pupil, and is generally displaced. It may be due to adhesions of the iris to the lens (posterior synechiæ) occurring in consequence of a fœtal iritis.

Discoria is a pupil abnormal in shape without a coloboma of the iris. It may be due to posterior synechiæ or persistence of a part of the pupillary membrane.

Polycoria is that condition in which there are two or more openings of the iris. As many as nine openings have been observed.

Irideremia, or aniridia, is the term applied to the condition in which there is an absence of the iris. It may be partial or complete, but is a bilateral abnormality. Aniridia is possibly due to a late separation of the lens and cornea, the entire anterior surface of the lens being involved.

On ophthalmoscopic examination, a large illuminated area representing the size of the lens is observed, and at times the fibres of the zonula can also be seen. By partially closing the lids some light is excluded, and this is the method the patient uses to exclude an excess of light. The vision is poor and nystagmus is often present.

Coloboma of the iris is the condition in which a part of the iris is deficient, causing an abnormality in the size and shape of the pupil. As the iris is a prolongation of the ciliary body, clefts in the choroid and ciliary body interfere with the development of the iris.

A coloboma might also be due to an abnormal adhesion between the lens and the cornea occurring during the embryonal stage, the defect of the iris representing the area of adhesion. An uncomplicated coloboma of the iris does not materially prejudice vision; when, however, a coloboma of the choroid, ciliary body, or lens is present, vision is more or less impaired.

Iridodonesis is the term that expresses a tremor of the iris. In buphthalmos, or in eyes with an ectopia or dislocation of the lens, the iris has not its normal support and is tremulous.

The **pupillary membrane**, which begins to disappear between the seventh and eighth months of fœtal life, is that part of the anterior fibrovascular sheath extending across the pupil. The anterior fibrovascular sheath consists of a meshwork of bloodvessels coming from the capsule of the lens: the periphery forms the anterior layer of the iris, the central portion the pupillary membrane. As the eye develops the hyaloid artery becomes smaller, and as the nutrition of the lens from this source is diminished the fibrovascular sheath disappears.

When for any reason a part of the sheath over the pupil does not disappear, the condition known as **persistent pupillary membrane** results. This abnormality is one of the most frequent of the congenital anomalies of the eye. It can be distinguished from posterior synechiæ by the fact that the pupillary membrane arises from the anterior surface of the iris.

Neoplasms.

Cystic tumors of the iris may contain a serous liquid or atheromatous material. They are very rare, and develop usually after operations whereby epithelium from the skin, conjunctiva, or cornea is carried into the wound. These cysts grow very slowly, and at the beginning may be removed by performing an iridectomy of the part involved. If left alone, they may obstruct the pupil, displace and opacify the lens, cause iritis and iridocyclitis, and, by increase of intraocular tension (secondary glaucoma), destroy the eye.

Sarcomas of the iris may be either pigmented or non-pigmented. They gradually fill the anterior chamber and finally perforate the eyeball. When very small, the excision of the involved part of the iris may be sufficient, but enucleation is the safest treatment.

Tuberculosis of the iris is a rare disease affecting tuberculous children. The tubercles may be disseminated or solitary.

Attention to the general health and measures to relieve the iritis constitute the *treatment* in favorable cases. In bad cases enucleation is indicated to remove the source of infection.

Injuries of the Iris.

Effects: Injuries whereby the iris is torn or lacerated are likely to produce *iritis*. When infection is carried into the eye through a wound involving the ciliary region or iris, an *iridocyclitis* is the result. *Simple contusion* of the bulb may cause only a *transient hyperæmia* of the iris; but *penetrating wounds* when *infected* may cause not only the *loss of sight* of the injured eye, but also *sympathetic inflammation* of the other.

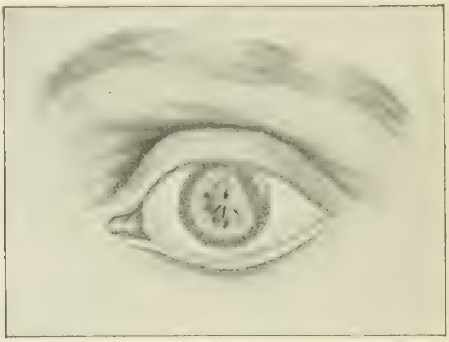
The **prognosis of traumatic iritis** depends upon the site of injury (the ciliary region being the most dangerous), the cleanliness of the wound, and the presence or not of foreign bodies in the eye.

In such conditions as are likely to produce sympathetic inflammation, the eye should be *enucleated*.

Iridoplegia and **cycloplegia** are terms designating respectively paralysis of the sphincter iridis and ciliary muscle. These conditions may occur in consequence of *contusion* of the eye, and from this cause are generally permanent. *Hyphæmia* (blood in the anterior chamber) is usually observed in such cases.

The *treatment* consists in rest and application of cold at the beginning, and later instillation of eserine if not contraindicated by an iritis.

FIG. 16.



Traumatic iridodialysis. (Jäger.)

Iridodialysis (Fig. 16) is the term that expresses a detachment of the iris from the ciliary body. The detachment varies in extent; it may be very small or the entire iris may be torn from the ciliary attachment. In partial detachment monocular diplopia may occur when two pictures are projected upon the retina, the one through the pupil, the other through the gap. Total detachment of the iris is called an *aniridia traumatica;* loss of a part of the iris, *coloboma traumatica.* In all cases of iridodialysis hyphæmia is present, owing to rupture of the vessels of the iris.

Treatment: The tendency to hemorrhage indicates the use of cold and observance of quiet. If *iritis* develop, atropine must be used.

In consequence of **penetrating injuries** of the eyeball a *cyst* in the iris-angle, due to an accumulation of aqueous in an inversion of the pectinate ligament and adjoining endothelium, is occasionally observed.

It should be removed before it occasions cyclitic irritation or increase of intraocular tension. A cyst may also occur in the *parenchyma* of the iris from like injury, and can be removed with the portion of the iris in which it lays.

Foreign bodies in the iris are rare, but chips of metal or stone have been known to become encysted and remain for years. Foreign bodies should be removed even if an iridectomy be necessary.

Iritis.

Definition: Iritis is an *inflammation of the iris* characterized by severe neuralgic pain about the orbit, photophobia, lachrymation, engorgement of the ciliary vessels, muddy appearance of the iris, sluggishness of the pupil, and more or less disturbance of vision.

Differential diagnosis: Iritis is frequently mistaken for *conjunctivitis;* and sometimes an *acute inflammatory glaucoma* is thought to be an iritis. Both mistakes are serious, and the latter may be disastrous.

In *conjunctivitis*, however slight, there is more or less mucous discharge, which glues the lids together. In *iritis*, however severe, the secretions are thin and the lids are never glued together. The use of atropine soon shows the existence of an iritis, in which case the pupil either does not dilate at all or yields slowly and imperfectly. When *posterior synechiæ* have formed, the pupil is irregular in shape.

In *iritis* the pupil is usually small; in *glaucoma*, large. In *glaucoma*, moreover, the tension is increased; there are anæsthesia of the cornea and a halo around lights. The use of atropine in glaucoma might destroy sight in a few days by increasing the intraocular tension. Hence the differential diagnosis between glaucoma and iritis becomes a very important consideration in the use of atropine.

Etiology: Iritis may be primary or secondary.

Primary iritis is sometimes caused by constitutional diseases, as syphilis, scrofula, tuberculosis, rheumatism, gonorrhœal rheumatism, acute infectious diseases, or diabetes. Traumatism and sympathetic inflammation are local etiological factors. An idiopathic iritis of which the cause cannot be determined may also occur.

Secondary iritis frequently complicates corneal diseases and scleritis; retinal detachment and choroiditis less often cause it.

The **symptoms** of iritis are pain, photophobia, lachrymation, conjunctival congestion, circumcorneal or ciliary redness, imperfect action of the pupil, exudation of serous, plastic, or purulent material into the parenchyma of the iris, upon its anterior or posterior surface, into the pupillary area or the aqueous, and impaired vision. One or more of these symptoms may be present, so that the diagnosis is usually not difficult. The use of atropine is an invaluable *diagnostic agent* in some cases: the slow and imperfect response of the iris to the mydriatic influence of the alkaloid and the irregularity of the pupil are pathognomonic.

An iritis may occur alone, but it at times involves the *ciliary body*, producing an iridocyclitis; in which case there may be present, besides the ordinary symptoms of an iritis, œdema of the lids, pain on pressure over the ciliary region, complete posterior synechia, impairment of vision, all out of proportion to the condition of the anterior chamber, or change of tension.

The **prognosis** of an iridocyclitis is more serious than that of a simple iritis, as might be inferred from the severity of the symptoms.

The **consequences** of an iritis or iridocyclitis which impair the usefulness of the eye are *exudates* and *synechiæ*.

Posterior synechiæ when small occasion little or no damage to sight, but when circular (seclusio pupillæ) the drainage of the nutrient liquid supplied by the ciliary processes from the posterior to the anterior chamber is interfered with, and the accumulation of the liquid in the posterior chamber causes a bulging and finally atrophic iris; and by increase of intraocular tension a secondary glaucoma results.

When the pupillary area is filled with *exudate* the sight is materially impaired. When an exudate imbeds the lens, from faulty nutrition, a cataract results; when the exudate displaces the vitreous, shrinking of the bulb (phthisis bulbi) follows, and sight is lost from detachment of the retina.

The **treatment** of iritis and iridocyclitis is constitutional, local, and surgical, according to the cause and complications.

The appropriate constitutional treatment should be instituted when syphilis, rheumatism, malaria, diabetes, gout, anæmia, scrofula, tuberculosis, or gonorrhœa seems to be a causal factor of the iritis.

Atropine should be employed in all cases, unless contra-indicated by increase of tension, to dilate the pupil until the inflammation has subsided. The atropine paralyzes the ciliary muscle and by dilating the pupil prevents the formation of adhesions to the lens and reduces the congestion of the iris and exudation. Light adhesions to the lens are frequently disturbed by timely use of atropine.

Pain and inflammation are reduced by the application of heat, either moist or dry, or of cold over the eye. The feelings of the patient are of importance in the selection of heat or cold, but heat will be generally found the more acceptable. The abstraction of blood from the temples by leeches (three or four will suffice) or by the artificial leech sometimes gives immediate relief; but this should only be practised in plethoric subjects.

Narcotics and the coal-tar preparations are sometimes indispensable in affording relief from pain.

When the signs of sympathetic iritis are slight, enucleation of the injured eye may save the other; but when the symptoms are well developed, no treatment is of use.

Paracentesis and iridectomy are the surgical procedures advocated for the relief of persistent increase of tension occurring during the acute stage of iritis or iridocyclitis. Paracentesis should first be tried, and if unsuccessful, an iridectomy may be thought of when the iris has not undergone great structural changes.

The *consequences* of iritis and iridocyclitis which call for treatment are small posterior synechiæ, the evidences of which are notching of the pupil; annular or circular posterior synechiæ (exclusio pupillæ); and total posterior synechiæ, in which the entire posterior chamber is filled with exudate. Small localized posterior synechiæ often yield to atropine and cocaine in combination. Eserine, followed by atropine, whereby the pupil is dilated after myosis has been produced, is a successful method in the smaller synechiæ, but more extensive adhesions resist all but operative treatment. An annular posterior synechia calls for operation as soon as the eye becomes quiescent, in order to restore the communication between the posterior and anterior chambers. In a simple case the iridectomy should be made in the upper segment; but when the pupil is occluded the lower nasal side should be selected in order to favor vision. A total posterior synechia requires an iridectomy, but since it is not generally possible to remove a piece of the iris sufficiently large, extraction of the lens will often be necessary. An iridectomy may be done when the lens is shrunken or absent.

Abnormalities of Movement of the Iris.

These include *mydriasis, myosis, anisocoria, iridodonesis,* and *hippus.*

Mydriasis, or dilatation of the pupil, may be spasmodic or paralytic. When spasmodic it may be a symptom of cerebral or spinal disease, or due to irritation direct or reflex of the cervical sympathetic.

Paralytic mydriasis is much more common, and is due to paresis or paralysis of the motor oculi; cerebral diseases affecting the nucleus of the third nerve; use of mydriatics, as atropine, homatropine, daturine, and scopolamine; poisoning by ptomaines or the products of the diphtheria bacillus; syphilis; pressure on the ciliary nerves from increase of intraocular tension; and injury producing iridoplegia.

The *prognosis* and *treatment* depend upon the cause. Mydriasis from injury, cerebral diseases, and glaucoma is often permanent.

Myosis, like mydriasis, may be spasmodic or paralytic. When spasmodic, the myosis is due to agents like eserine and pilocarpine; when paralytic, to paralysis of the cervical sympathetic and to spinal disease, as tabes.

Anisocoria is a term which designates a difference in the size of the pupils. In young nervous individuals, mydriasis, seldom complete, may occur, but the condition is temporary, although subject to relapses. Syphilis is the more common cause of unilateral mydriasis, and yields to treatment.

Cycloplegia, or paralysis of the accomodation, sometimes complicates the condition.

Iridodonesis, or pathological trembling of the iris, occurs when the iris, because of the absence of the lens, has not the proper support.

Hippus is a condition in which the size of the pupil is subject to rapid variations because of clonic or intermittent spasm of the iris. It occurs in various nervous diseases, both organic and functional.

THE CILIARY BODY.

Anatomy.

The **ciliary body** connects the choroid and the iris, forming the middle zone of the uveal tract (Fig. 17).

It consists of the *ciliary muscle*, composed of unstriated muscle-fibres, *connective tissue*, *bloodvessels*, and *nerves;* and the *ciliary processes*, which are similar in structure to the choroid, with the exception that the arteries empty directly into the veins without the intervention of capillaries.

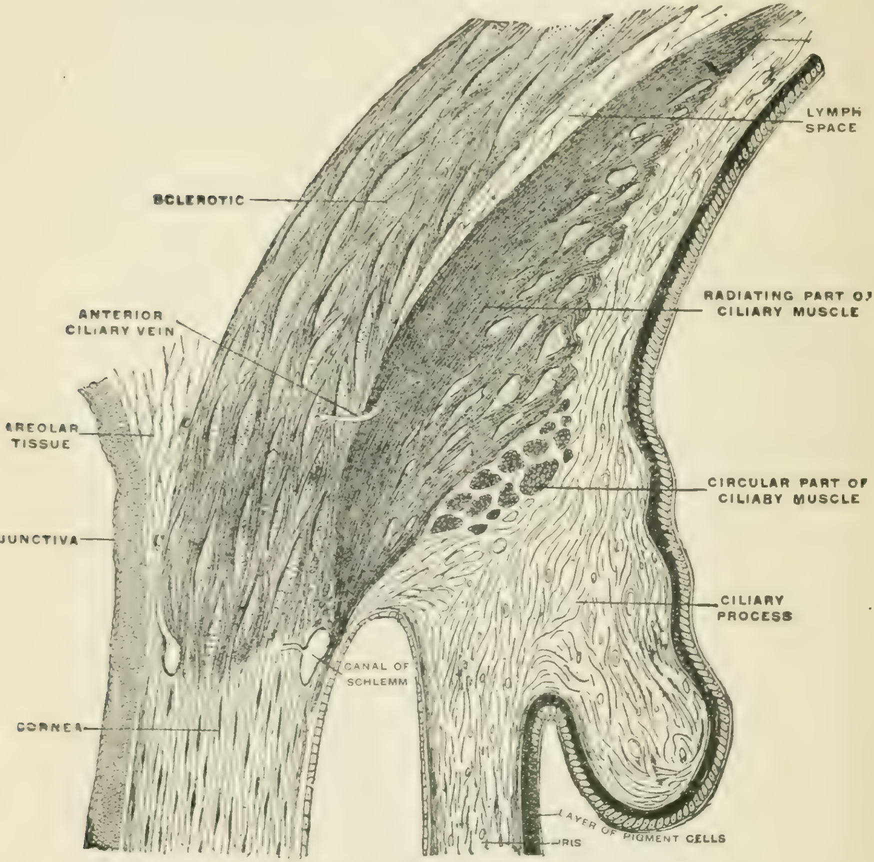
The *inner surface* of the ciliary body is a continuation of the pigment-layer of the retina, and is known as the **pars ciliaris retinæ.**

Anteroposteriorly the ciliary body is about one-quarter of an inch in width, and a vertical section of the bulb shows that it is triangular in shape, the base being directed forward and the apex merging into the choroid.

The **ciliary muscle** consists of longitudinal, radiating, and circular fibres, arranged in this order from without inward. The **ciliary processes** represent the choroid and the pigment-layer of the retina: the former is external, the latter internal. The processes anteriorly are thrown into from seventy to eighty folds, the posterior portion is perfectly smooth.

The *ciliary muscle* is the means of effecting *accommodation* for vision at different distances. When the eye is at rest, implying rest of the ciliary muscle, the lens is flattened by the tension of the suspensory ligament, which is produced by the position of the ciliary body. When the ciliary muscle is at rest, the ciliary body is at a greater distance from the equator of the lens than during accommodation, and the anterior border of the ciliary body during rest extends farther backward. The position of the ciliary body being such that the tension of the suspensory ligament is increased during rest of the ciliary muscle, it will be evident that the lens is

Fig. 17.



Meridional section of the eye in the region of the ciliary muscle. (Testut.)

flattened from the tension of the suspensory ligament when the eye is at rest. When the tension is removed because of the change of position of the ciliary body, which is nearer the lens during accommodation, the natural elasticity of the lens increases its convexity. During accommodation the entire iris moves forward and the pupil is contracted.

The *ciliary processes* supply nutrition to the lens and contiguous portion of the vitreous, and replenish largely the loss of the aqueous humor.

The ciliary muscle is *supplied* by fibres from the third nerve, which also supplies the sphincter pupillæ and internal recti muscles. The intimate connection of these fibres explains the convergence of the eyeball and contraction of the pupil which are associated with accommodation.

Anomalies and Neoplasms.

Coloboma of the ciliary body is due to a persistence of the cleft, and is often associated with a coloboma of the iris, choroid, or lens. Inasmuch as the iris grows from the anterior border of the ciliary body, a coloboma of the ciliary body is the cause of the coexisting coloboma of the iris.

Neoplasms: New growths, excluding tubercle and gummata, are very rare. When present, enucleation is the *treatment.*

Injuries.

Wounds of all kinds may involve the ciliary region, and, because of the danger to the uninjured eye from sympathetic inflammation, are of the greatest importance.

Injuries of the choroid or iris from a *foreign body* are very serious from the possibility of causing sympathetic trouble. But if the ciliary region be involved the danger is the greatest. The nearer the wound is to the ciliary region the greater becomes the danger.

A *sympathetic inflammation* is usually the **result of trauma** affecting the iris, ciliary body, or choroid, whereby, after a variable time (from a few weeks to many years), a plastic or serous uveitis occurs in the other eye.

The most popular theory explaining the production of sympathetic inflammation is the one which teaches that infection is conveyed to the other eye along the optic nerve and its sheaths.

When an injured eye some time after receipt of the injury becomes painful and the other presents signs of irritation, such as photophobia, lachrymation, congestion, or tenderness, sympathetic inflammation should be thought of. Usually it will be noticed that the accommodation of the uninjured eye is crippled. The ciliary body is involved at first, then the iris, and finally the choroid.

The **treatment** of sympathetic inflammation is most satisfactory when prophylactic. An injury in the ciliary region, the eye being sightless, is best treated by an enucleation. When a panophthalmitis occurs in consequence of traumatism, the danger of sympathetic inflammation is not great.

When sympathetic inflammation has fairly developed, treatment is useless. If any vision remains in the injured eye after marked signs of sympathetic ophthalmitis it should not be enucleated, since the sight of the injured eye is then apt to be all the patient will possess. If some time after injury to an eye a patient presents himself with tenderness and painfulness of that eye, it is well to suggest an enucleation as the only treatment.

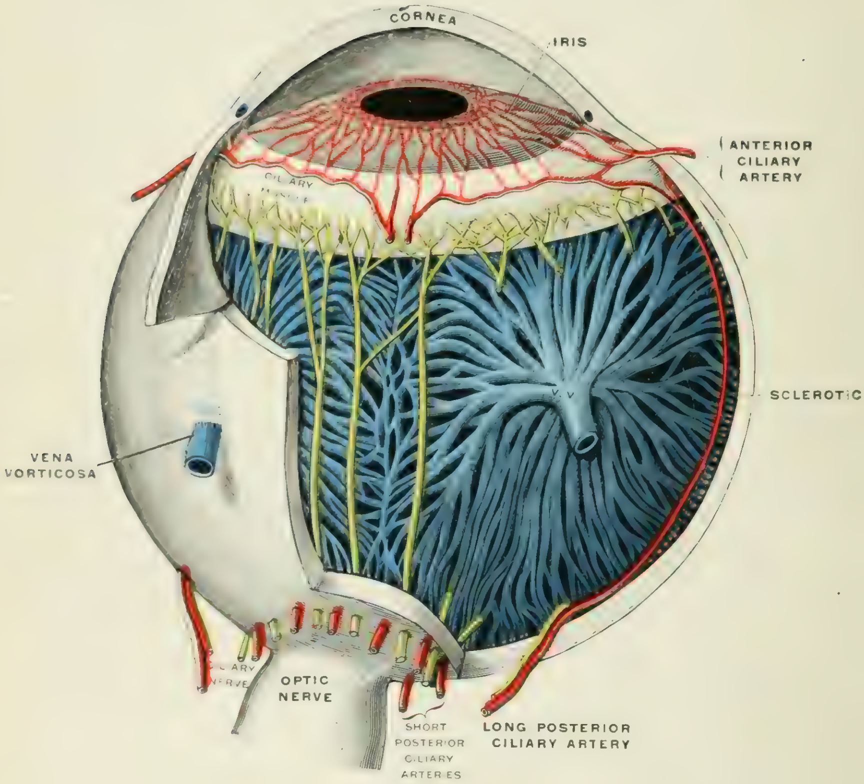
In consequence of injury, cycloplegia or paralysis of the ciliary muscle often accompanies iridoplegia. Cycloplegia from traumatism is usually permanent.

Diseases of the Ciliary Body.

An **inflammation** of the ciliary body—*cyclitis*—seldom occurs alone, the iris being almost invariably affected. An iritis may occur without involving the ciliary body, but sometimes the ciliary body becomes secondarily affected. An *iridocyclitis* is an inflammation of both the iris and ciliary body.

A **simple cyclitis** presents many symptoms of an iritis—pain, photophobia, lachrymation, ciliary redness, change of color of the iris, impairment of sight, and evidences of exudate; but besides these, a *hypopyon*, which rapidly appears and disappears, a *deposition of particles* upon the posterior surface of the cornea (constituting keratitis punctata), tenderness over the ciliary region, and increase of intraocular tension occur. When the cyclitis is due to sympathetic inflammation, a whitening of the eyebrows is sometimes observed. A simple

PLATE IV.



Vessels and Nerves of the Choroid and Iris, seen from above. The Sclerotic and Cornea have been largely removed. (Testut.)

cyclitis is usually a chronic trouble, and is subject to frequent exacerbations.

The **causes** of cyclitis are the same as those of iritis, and the **treatment** is likewise the same.

THE CHOROID.

Anatomy.

The *choroid*, ciliary body, and iris form the middle coat of the eye, and are known as the **uveal tract.**

The **choroid**, forming the posterior seven-tenths of the middle tunic, extends from the entrance of the optic nerve to the ora serrata, where the ciliary body begins. It is loosely attached to the sclera, except at the margin of the disk, where there is a ring of connective-tissue fibres. The choroid is from $\frac{1}{250}$ to $\frac{1}{150}$ of an inch in thickness and consists largely of bloodvessels (Plate IV.).

The lamina fusca, tunica vasculosa, membrana choriocapillaris, and membrana limitans are the **four layers** of the choroid.

The **lamina fusca**, or suprachoroidea, consists of loose cellular connective-tissue fibres, among which are imbedded many pigment-cells, lymph-corpuscles, and elastic fibres. This layer loosely connects the choroid and the sclera, between which is the perichoroidal lymph-space, which communicates with the lymph-space in Tenon's capsule.

The **tunica vasculosa** contains the ramifications of the long and short posterior ciliary arteries, which are internal to the veins. These arteries empty into the choriocapillary layer, and the blood from this layer and much from the ciliary body and iris leave the eye by the *vorticose veins*, of which there are from four to six. The smaller veins empty into larger ones, which finally form a single large vein, or *vena vorticosa*, which is the summit or vortex of each group or set of veins.

The **membrana choriocapillaris** contains the capillary system formed by the long and short posterior ciliary arteries.

The **membrana limitans**, or *lamina vitrea*, is very thin, and is firmly attached to the membrana capillaris. The pigment-layer of the retina covers the inner surface of this layer.

The choroid and ciliary processes furnish **nutritive lymph** to the lens, anterior part of the vitreous, and external layers of the retina.

Congenital Anomalies of the Choroid.

Albinism: In cases of complete albinism, the choroidal and retinal pigment is wanting, and the choroidal vessels can be easily seen upon a light background with the ophthalmoscope. The pupil presents a pinkish reflex. The visual acuity is always diminished, and the eyeballs make rapid oscillations, a condition known as nystagmus. Myopia and strabismus often complicate albinism.

Coloboma: Absence of the choroid usually occurs in the choroidal fissure, but may occur in the area of the macula. With the ophthalmoscope, an oval whitish area is seen extending from the lower margin of the disk toward the ciliary body, the long diameter being that of the anteroposterior axis of the globe. Because of the absence of the pigment and the choroidal vessels the white sclera is seen, representing the size of the defect. When the retina is perfect, its vessels are seen to cross the area of defect; when absent the vision is all the worse.

Coloboma in the choroidal fissure is often accompanied by similar defects of the ciliary body and iris, as well as other anomalies of development. A small crescentic defect of choroid at the inferior margin of the disk simulates the myopic crescent in appearance, but not in location. This is known as the coloboma of Fuchs.

A coloboma of the choroid about the disk may or may not be associated with a coloboma of the nerve-sheath.

Colobomas are often hereditary, and occur in individuals having other congenital malformations.

The size and position of the coloboma and the condition of the eye determine the impairment of vision.

Treatment is useless.

Neoplasms.

Sarcoma, usually a melanosarcoma, is a very rare growth of the choroid, occurring most frequently between the ages of forty and sixty. Since *glioma* of the retina occurs exclusively in children, the patient's age is a diagnostic point of value, inasmuch as both neoplasms run a similar course.

Symptoms: The *first stage* of the growth can be diagnosed only with the ophthalmoscope, more or less detachment of the retina and corresponding disturbance of vision being present.

The *second stage* is that in which a secondary glaucoma or, more rarely, an iridocyclitis results.

The *third stage* is characterized by perforation of the bulb, and the *fourth* by the occurrence of metastatic nodules in other organs.

The **prognosis** is bad as regards life, for most patients succumb unless the malignant neoplasm be removed at the beginning.

Treatment: At times enucleation does not suffice, and an exenteration of the orbit, including the periosteum, is necessary.

Injuries.

Contusion may produce a **rupture** of the choroid, which is either a simple condition or a complication of other injuries from the same cause. Immediately after the injury the diagnosis may be difficult owing to the hemorrhage.

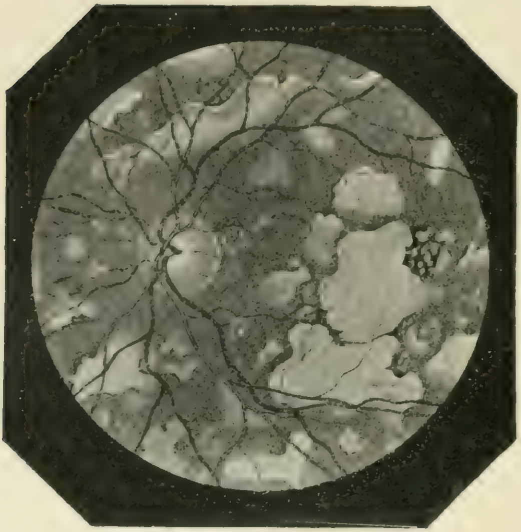
The rupture usually appears like a rent or streak, which is sometimes forked and is often concentric with the optic pupilla. Unless the rupture has involved the retina, the retinal vessels will be seen to pass over the rent. The location of the rupture determines the danger to vision, the macular region being the worst.

Treatment consists in rest, atropine, and antiphlogistic measures, as use of cold cloths and local bloodletting.

Detachment of the choroid sometimes occurs, but is not recognized, owing to the opacity of the media, until the eye has been enucleated. When the choroid is injured, in consequence of a **penetrating wound**, the condition is very bad, particularly when infection has taken place.

Foreign bodies which injure the choroid in their course should be removed on general principles.

FIG. 18.



Syphilitic choroiditis. (Jäger.)

Injuries producing a **suppurative choroiditis** lead to panophthalmitis and phthisis bulbi, with loss of sight and shape of the eye.

CHOROIDITIS.

The choroid is subject to three kinds of inflammations: tubercular; an exudative or plastic; and a suppurative.

Tuberculosis of the choroid. The nodule may be solitary; or multiple and disseminated.

When *solitary*, a very rare condition, the course of the disease is that of a neoplasm, and most often occurs in youth. The eye is invariably lost, but the patient's life may be saved by an *enucleation* if the eye is the single and primary seat of the affection.

Disseminated tubercular nodules, usually less than ten in number, are a manifestation of miliary tuberculosis, and when seen with the ophthalmoscope assist in diagnosis of the same. The nodules can be differentiated from other inflammatory choroidal changes (choroiditis disseminata) by their rapid growth and absence of pigment.

Exudative Choroiditis.

Pathology: It is manifested by the formation of an exudate at the site of inflammation. Absorption of the exudate leads to atrophy of the choroid and development of pigment, which more often accumulates at the margins of the diseased spots. In some cases the choroid alone is diseased, but frequently the retina is more or less involved, in which case the condition is called a *choroidoretinitis.*

An exudative choroiditis does not, as a rule, involve the iris and ciliary body as does the suppurative form, and the media remain transparent, so that the diagnosis with the ophthalmoscope is an easy matter. An exudative choroiditis may affect one or both eyes, and the **cause** is often syphilis, either hereditary or acquired; scrofula, anæmia, and diseases which lower the vitality.

According to the location and arrangement of the lesions, the implication of the retina, or the cause, exudative choroiditis is **classified** as follows: *choroiditis* or *choroidoretinitis disseminata; choroiditis areolaris; choroiditis centralis; choroidoretinitis specifica*, and *staphyloma posticum*, or posterior sclerochoroiditis.

Choroiditis disseminata is characterized by a number of spots which are scattered about the fundus, the papilla representing the centre. The atrophy of the choroid permits the view of the sclera, so that the spots are yellow or white according to the degree of atrophy. The disease usually begins at the periphery and extends toward the papilla. Sometimes the lesions are confined to the periphery, the papilla and circumpapillary portion of the choroid showing evidences of atrophy. These cases usually develop in syphilitic subjects. In some old cases of disseminated choroiditis the retina becomes involved, so that the deposition of pigment may cover the retinal vessels, which, because of a diminution in size, indicates an atrophy of the retina. The papilla is often blurred, the margins being indistinct and the cupping not demonstrable. Such cases receive the name *choroidoretinitis.*

Choroiditis areolaris presents for examination lesions of which the macular region forms the centre. The lesions form about the macula in such a way that the recent spots are the more distant.

Choroiditis centralis has a lesion which covers the macular region, producing a central scotoma or blind spot. High degrees of myopia, syphilis, and contusions may produce it.

Choroidoretinitis specifica is due to syphilis (Fig. 18), and involves the retina and choroid, as the name implies. The vitreous and retina become less transparent, and in the vitreous small dust-like particles develop. A papilloretinitis, characterized by a haziness of the papilla and circumpapillary portion of the retina, is usually present. Atrophy of the retina with its vessels notably diminished in size and a deposition of pigment are the results of this disease.

A **posterior staphyloma** is that condition of the choroid characterized by an atrophy adjacent to the disk associated with a bulging of the sclerotic, which is greatest at the temporal border of the disk. This condition is the cause of malignant forms of myopia and often complicates myopia of high degree.

The **treatment** of all forms of exudative choroiditis depends upon the cause. Syphilis and the various dyscrasias demand their particular treatment. To facilitate absorption of the exudates, the iodides and diaphoretics (as pilocarpine and the salicylates) are indicated. Absolute rest of the eye and a darkened room are necessary for some cases.

Suppurative Choroiditis.

Etiology: This disease is due to infection, the source being either within the patient—endogenous—producing the disease by metastasis; or from without—exogenous. Phlegmonous inflammation of the orbit, meningitis, and pyaemia are the more common sources of endogenous infection. Traumatism and spontaneous perforations of the cornea in consequence of ulcers or abscesses may produce infection from without.

Symptoms: This disease is associated with secondary inflammation of the ciliary body and iris, and the symptoms are, therefore, intense. The lids are swollen, the conjunctiva is congested and œdematous, the cornea dull, the aqueous is no longer transparent, and a small amount of pus settles at the bottom of the anterior chamber—hypopyon. The iridocyclitis produces great pain and tenderness. The choroidal exudate which occurs between the choroid and retina extends into the cavity of the vitreous. The intraocular pressure is increased because of the exudate and possible interference with drainage, due to a seclusio pupillæ in the first stages. In less severe cases the globe begins to shrink, absorption taking place: *Atrophy;* in severer cases the globe becomes an abscess—panophthalmitis, and the pus finally finds an exit through the sclera, leading to a condition known as *phthisis bulbi.*

Diagnosis:

Phthisis bulbi.	*Atrophia bulbi.*
Follows panophthalmitis, and the eyeball is diminished in size from escape of some of the contents through an opening.	Follows iridocyclitis, iridochoroiditis serosa, or suppurative choroiditis (not severe). The eyeball becomes smaller from absorption of exudate.
The contents of the eye are more or less destroyed or lost, the choroid in particular. Sympathetic inflammation is not so likely to occur, and enucleation, therefore, is not often necessary.	The contents of the eye are not necessarily much damaged, and sympathetic inflammation is possible. Enucleation of the eye is frequently called for, to prevent sympathetic inflammation and relieve pain.
Eyeball is considerably reduced in size in a short time.	Eyeball gradually diminishes in size.
Artificial eye may be used.	Artificial eye contraindicated.

When an *exudate*, the result of choroiditis, can be seen behind the lens in children under two years of age, the condition is named *pseudoglioma*, from the possibility of mistaking it for a glioma.

The **treatment** of suppurative choroiditis is palliative. To relieve the pain, heat and anodynes are useful. An enucleation, if performed in the early stages, is not dangerous, and much suffering is forestalled. An incision may be made through the sclera and evacuation of the pus thus hastened. In the later stages an enucleation is dangerous, since a suppurative meningitis may result.

GLAUCOMA.

Definition: *Glaucoma* is a disease of the eyeball, characterized by an increase of tension.

Varieties: As regards its duration, it may be *acute*, *subacute*, or *chronic*; and it is either *primary*, when there has been no antecedent causal disease; or *secondary*, when the increase of tension is due to some previous ocular affection.

Primary glaucoma may occur with or without inflammatory manifestations, and is accordingly called either *inflammatory* or *simple*. *Hydrophthalmos* is a disease of childhood, characterized by an extraordinary size of the eyeball and an increase of tension which is not accompanied by inflammatory reaction: it is therefore a form of simple glaucoma.

Normal tension of the eyeball is expressed by the letter T; *increased tension* (hypertony), according to the degree, by T + 1, T + 2, or T + 3; *reduced tension* (hypotony), according to the degree, by T − 1, T − 2 or T − 3. T + 1 ? signifies doubtful increase of tension, and T − 1 ? doubtful reduction of tension. To determine the tension, palpation with the two index-fingers, the hands resting on the patient's face, is a ready method.

Primary Glaucoma.

Primary glaucoma has a doubtful **etiology**, but several conditions are observed in eyes blinded by glaucoma which explain the increase of tension.

In order to interpret these conditions, the *drainage* of the aqueous chamber must be understood. The nutrient fluid secreted by the ciliary body to nourish the vitreous and the lens and replenish the aqueous chamber passes through the pupil into the anterior chamber and escapes from the eye by filtration through the pectinate ligament into Schlemm's canal and the venous system. The angle formed by the iris, the pectinate ligament, and the posterior wall of Schlemm's canal is known as the *filtration-angle;* and it is evident that any condition which interferes with or obliterates this angle will prevent the drainage of the eye, whereby an accumulation of the aqueous will occur, and an increase of intraocular tension is the inevitable consequence.

In cases of primary glaucoma the filtration-angle is either closed or considerably compressed, and the iris is either adherent to the cornea and pectinate ligament or pressed against them. In recent cases the ciliary processes and the ciliary muscle are drawn forward, but later they are atrophied and retracted.

The obliteration of the filtration-angle is due primarily to pressure of the ciliary processes against the iris, and the explanation of increased tension is the retention of the intraocular fluid.

Acute primary inflammatory glaucoma is bilateral, one eye and then the other being affected, and occurs almost exclusively in individuals past forty years of age.

Etiology: The eyes are frequently hypermetropic; sex does not predispose; heredity is often demonstrable; the Jewish race is predisposed; mental emotions are sometimes causal; sleeplessness, great and prolonged pain, starvation, respiration of foul air, excessive mental application, vomiting, and febrile diseases may lead to a depressed state of the body which favors glaucoma; myotics abort light cases and relieve severe ones, but do not prevent relapses; iridectomy restores normal tension and a sclerotomy is a palliative measure; an eye enucleated because of inflammatory glaucoma remains hard after enucleation; and the examination of an eye between the attacks of light glaucoma shows no pathological changes. The above facts represent what is positively known about acute inflammatory glaucoma.

Symptoms: Acute primary glaucoma is sometimes preceded by premonitory symptoms, as dimness and rainbow vision; but the onset may be sudden and violent in the fulminating form. After some exciting cause, the eye becomes painful and a neuralgic pain radiates from the affected eye over the temple and side of the head. The lids are swollen and the conjunctiva is congested. The cornea becomes "steamy" from œdema of its tissues, is not sensitive, and vision is much impaired. The pupil is usually dilated and does not react, and accommodation is diminished. The tension of the eyeball will be found more or less increased, and the anterior chamber becomes more shallow. Ophthalmoscopic examination, when possible, shows that the arteries are smaller and the veins engorged. Hemorrhages in the retina and choroid are occasionally observed. A change of refraction of some dioptres is also observed in glaucoma. If *treatment* is not instituted, the sight is lost.

Subacute primany glaucoma is characterized by a recurrence at intervals of imperfect and rainbow vision at the beginning; but later the increased tension is permanent, and its results, the glaucomatous cupping of the disk and contraction of the visual field due to insufficient nutrition of the retina, take place. Blindness is the result unless operative interference comes to the rescue, when some vision is still retained.

Absolute glaucoma implies that the eye is sightless, the tension is excessive, and the disk excavated. The cornea is transparent but insensitive, the iris reduced to a narrow band, and the anterior chamber is very shallow. Absolute glaucoma leads to ectasias of the sclera, particularly the equatorial form, opacity of the lens, and a haziness of the cornea. These manifestations are those of glaucomatous degeneration, which is often accompanied by pain. The final outcome may be atrophy of the bulb, or perforating abscess of the cornea, with its consequent iridocyclitis or panophthalmitis with phthisis bulbi.

Glaucoma simplex is a chronic primary affection, the only symptom of which is impairment of the sight. Increase of intraocular tension is not present at all times, and may be absent altogether. Chronic primary glaucoma, as it is also called, develops slowly and insidiously, and may require many years to destroy the sight. Like inflammatory glaucoma, it affects both eyes, one shortly after the other. Since pain and inflammation are not present, the patient may not be aware of the disease until the visual field becomes considerably contracted, the central vision remaining good for a long time. The eye may appear normal even when blind, or an inflammatory glaucoma may supervene with its results.

Hydrophthalmos, or *buphthalmos*, occurs either congenitally or shortly after birth, and since the sclera at this period of existence is elastic, an increase of intraocular tension may increase the size of the eyeball. In adults the lamina cribrosa is the only part of the sclera which yields to pressure, and this accounts for the presence of the glaucomatous excavation or cupping. Hydrophthalmos may come to a standstill, in which case the tension becomes normal and some sight is retained; or it may progress until the eye reaches frightful dimensions and sight is lost. Hydrophthalmos is usually a bilateral disease.

The **treatment of primary glaucoma** is palliative and operative. Physostigmine (better known as eserine) and pilocarpine, because of the myosis they produce, increase the permeability of the filtration-angle and reduce the intraocular pressure. Cocaine dilates the pupil, but contracts the ciliary bloodvessels and diminishes the sensibility of the ciliary nerves, whereby the intraocular pressure is reduced. Cocaine in combination with physostigmine, the myotic action of which should dominate, is also useful. Atropine must be mentioned to be condemned in the treatment of primary glaucoma. It dilates the pupil, increases the intraocular pressure, and may not only precipitate an attack of glaucoma in a predisposed individual, but even destroy the sight in a short time when used during the disease. However valuable atropine is in the treatment of corneal and iritic disease, it is dangerous and disastrous in primary glaucoma.

Morphine, rest, warm nourishment, laxatives, and the local use of cold are valuable palliative measures in certain cases. The myotics are useful when the tension of the eye is notably increased, but of questionable utility in chronic glaucoma (simplex), which may not be accompanied by increase of tension.

An iridectomy, sclerotomy, and enucleation are the *operations* suggested for the relief or cure of primary glaucoma.

An *iridectomy* in order to be potent should be made in such a way that a large segment of iris is removed as near as possible to the ciliary attachment. The effect of a properly performed iridectomy upon the course of an acute inflammatory glaucoma is all the better for vision the sooner it is performed. An iridectomy is beset with some difficulty during this condition, owing to haziness of the cornea and shallowness of the anterior chamber, but its effect is sometimes curative. In chronic inflammatory glaucoma the result of the iridectomy will depend upon the duration of the cupping and atrophy of the optic nerve. Simple glaucoma is not so much benefited by an iridectomy, the most that can be expected being a retention of vision equal or nearly equal to that at the time of operation. Some cases actually do worse, and the eye is blinded after the iridectomy: these are the malignant ones.

A *sclerotomy*, whereby an opening is made through the sclera into the anterior chamber, may be performed in place of the iridectomy, but is not so reliable.

An *enucleation* is indicated in absolute glaucoma when, on account of atrophy of the iris, an iridectomy cannot be performed or would be useless to relieve the pain.

Hydrophthalmos is subject to the same treatment as the other primary forms of glaucoma; but an iridectomy may be serious from the escape of the vitreous through the suspensory ligament, which is apt to tear when the anterior chamber is emptied.

Secondary Glaucoma.

Definition: The increase of tension is due to some ocular disease which causes retention of the intraocular fluid.

Etiology: An annular posterior synechia, by preventing communication between the posterior and anterior chambers, pushes the iris forward and the filtration-angle is no longer permeable. Corneal wounds or ulcers when associated with anterior synechia interfere with the filtration-angle according to the amount of iris adherent to the cornea. When the cornea is perforated, establishing a fistula, the intraocular pressure is not increased so long as the aqueous can leak out of the corneal opening; but closure of a fistula of long duration may cause increased tension. A serous cyclitis, from the altered character of the secretion, may cause an accumulation of morbid fluid which is too thick to filter through the pectinate ligament in the anterior chamber, the result of which is a deepening of the anterior chamber and widening of the filtration-angle. After cataract operations secondary glaucoma may develop at a variable period of time. The filtration-angle is always found to be abnormal. A dislocated lens may produce increased tension; and a discission or wound of the lens, because of the swelling of the lens, may have a similar effect. Intraocular tumors at a certain stage produce intraocular pressure, and the filtration-angle will be found disturbed in these cases. Exudations in consequence of iridocyclitis or choroiditis sometimes lead to increase of tension, and hemorrhages into the vitreous may have a like result. Ectasia of the sclera is at times responsible for increase of tension.

The **treatment of secondary glaucoma** depends, of course, upon the cause. An annular posterior synechia requires an iridectomy to establish communication between the posterior and anterior chambers. Corneal staphyloma may be treated by incising the bulging part. Ectasias of the sclera producing glaucoma are best treated by an iridectomy if it be possible; otherwise by enucleation when the pain is considerable. The tension due to a serous cyclitis is relieved by a paracentesis repeated several times if necessary. A dislocated or swollen lens should be extracted when causing glaucoma if it be possible. Tumors require enucleation of the eye; and hemorrhagic glaucoma is usually not amenable to other treatment. All sightless and painful eyes indicate enucleation.

THE LENS.

Anatomy.

The **crystalline lens** is a transparent, lenticular body, biconvex in shape, the posterior curvature being the greater.

It is enclosed in an elastic envelope, or **capsule**, the anterior part being more than twice as thick as the posterior, and hence more subject to pathological conditions. The anterior part of this envelope is known as the *anterior capsule*, the posterior part as the *posterior capsule*, although the envelope is the same in structure. It is to the elasticity of the capsule that the lens owes the property of changing its curvature, whereby the rays of light may be brought to a focus on the retina, in adaptation of the eye for sight at different distances.

Equator and axis: The *equator* of the lens represents the margin at which the lenticular surfaces meet. The anteroposterior *axis* of the lens extends from the so-called anterior to the posterior pole, which is at the junction of the anterior and middle thirds of the optical axis of the eye.

The lens is held in place by the **suspensory ligament,** or zonula, whose lenticular attachments are, in front to the anterior capsule near the equator, behind to the corresponding margin of the posterior capsule.

The annular space, triangular on section, bounded by these anterior and posterior lamellæ of the zonula and the equatorial margins of the lens, is the *canal of Petit* of the older anatomists.

Accommodation: The lens and zonula separate the aqueous and vitreous humors. When the zonula exerts traction on the capsule the convexity of the lens is diminished; when it is relaxed the convexity is increased. These changes of the lens constitute accommodation. The lens up to adult life has throughout the same consistence. The central part, known as the nucleus, then becomes denser than the external, or cortical, part. With increasing age, the lens-substance becomes less pliable, and on this account the elasticity is less and the power of accommodation diminishes.

The **substance of the lens** consists of lens-fibres, each of which is composed of two layers of epithelium. These ribbon-like fibres are so arranged that a part of each is on the anterior as well as the posterior surface. They are kept in apposition by a cement-substance, and the lines of apposition constitute the lens-stars, which can be better observed in young subjects than in adults.

The *lens-stars* on the anterior surface of the lens resemble an inverted Y; those on the posterior surface the erect letter Y. The lens-fibres forming the inner and middle portions of the lens have serrated edges; the peripheral lens-fibres are smooth and have nuclei. The *minute spaces* between the fibres contain the semi-fluid cement-substance, and these channels afford passage-ways for the transmission of the nutritive fluid to the fibres of the lens. The ciliary body secretes the fluid for nutrition of the lens, and this enters near the equator.

The **substance of the suspensory ligament,** the zonule of Zinn, or the zonula ciliaris, is composed of transparent homogeneous connective-tissue fibres, which arise largely from between the folds of the ciliary processes and also from the ora serrata and the smooth surface of the ciliary body. The fibres which arise posteriorly are inserted on the posterior surface of the lens; the others cross in such a way that the more anterior fibres are inserted on the posterior surface and the posterior fibres go to the anterior surface of the lens.

In **accommodation**, the elastic lens, the suspensory ligament, and the ciliary body are concerned. When the eye is accommodating for the near point, the lens, because of its elasticity, assumes its greatest convexity, the suspensory ligament being slack from the position of the ciliary body, which during this act moves forward and inward. When the ciliary muscle is at rest, the ciliary body makes traction upon the fibres of the suspensory ligament, and in consequence of this the convexity of the lens is diminished and the eye is adapted for distant vision.

Malformations.

Congenital absence of the lens (aphakia) is very rarely seen, and then mostly in microphthalmic eyes.

Lenticonus is a rare anomaly in which the anterior surface (less often the posterior surface) presents a conical projection. After the use of a mydriatic, an ophthalmoscopic examination reveals a central red circular area surrounded by a narrow ring-like shadow, external to which is an illuminated zone.

Coloboma of the lens sometimes is associated with a congenital coloboma of the iris. The lens is more or less defective at the site of the coloboma iridis.

Ectopia lentis congenitum (subluxation of the lens) is often hereditary. It is due to a difference of width of the suspensory ligament, whereby the lens is not held in proper position. A congenitally dislocated lens is sometimes cataractous at birth or may become so later. A partial dislocation very often becomes a complete one, when the iris will be found tremulous. The *treatment* depends upon the patient's vision, operation (removal of the lens) being only undertaken when other measures (those for correcting refraction) are unsuccessful. When the dislocated lens is producing irritation and cannot be removed, enucleation is indicated.

Congenital cataracts involve either the entire lens or a portion of it.

Complete cataracts may be fluid, gelatinous, or hard in consistency. Discission is the treatment, and this may be done when the patient has lived three or four months. In hard and shrunken cataracts, discission is not successful, since the membrane remaining after operation always remains opaque.

The *partial cataracts* which may be congenital are *anterior polar*, *posterior polar*, *perinuclear*, and *dotted*.

Dotted cataracts are apparent as small spots or streaks in the cornea, and are often hereditary. The dots occur mostly in the periphery of the lens, and do not occasion any disturbance of vision: therefore they require no treatment.

A *posterior polar cataract* is an opacity of the posterior capsule at the central part, due to the presence of the hyaloid artery, which extends across the vitreous chamber in fœtal life. No *treatment* is called for.

The other forms of partial cataract will be considered under *Cataracts*.

Injuries.

A **traumatic dislocation** of the lens usually occurs as a result of contusion of the bulb. In some extensive ruptures of the bulb the lens may be expelled with other of the contents and the eye is usually lost.

Treatment: When suitable glasses assist the vision, these should be prescribed. When symptoms of secondary glaucoma or iridocyclitis occur, the lens should, if possible, be extracted. This is easy when the lens is in the anterior chamber, difficult or impossible in a partial dislocation, and quite impossible when the lens floats in the vitreous. When removal of the lens is not practicable, an iridectomy may relieve the tension or the inflammation. When a sightless eye is painful and inflamed because of a dislocated lens, an enucleation relieves the patient and prevents a possible sympathetic inflammation.

Traumatic cataracts are due to an infinite variety of injuries. Simple concussion, lightning-stroke, penetrating wounds of the bulb, associated or not with concussion, or the entrance of a foreign body into the eye may produce cataracts.

The **prognosis of injuries to the lens** depends upon the presence of a foreign body in the eye, infection, iridocyclitis, secondary glaucoma, and the situation and extent of the wound.

Treatment of injuries: A traumatic cataract may be treated by discission or extraction, according to the patient's age. When the lens-capsule is torn, the lens-substance should be left alone and allowed to absorb, unless its presence indicates danger by irritation or increase of tension, when extraction is indicated.

When traumatic cataracts are not complicated by wounds of the ciliary region or the sclera, detachment of the retina, rupture of the choroid, or iridocyclitis, the removal of the lens offers good hope for vision. When foreign bodies enter the eye and remain imbedded in the lens, the removal of the lens should be undertaken as soon as it is ripe. An involvement of the iris to any extent is prejudicial to success in the management of a traumatic cataract. A prolapsed iris should be cut off. The integrity of the capsule need not necessarily be impaired in traumatic cataracts, yet in most cases laceration of the capsule, exposing the lens-substance to the action of the aqueous, occurs.

Cataracts.

Definition: An opacity of the lens or of its capsule, or both, is known as a cataract.

The following **kinds** of cataract, classified by the location of the opacity, are seen: anterior polar; posterior polar; zonular; anterior and posterior cortical; and nuclear.

Besides these, senile, congenital, secondary, Morgagnian, and traumatic cataracts occur.

All cataracts are partial or complete, or stationary or progressive.

The **anterior polar cataract** is located between the anterior capsule and the lens at its anterior pole, and appears like a small white spot. It may be congenital or acquired.

When *congenital*, it is bilateral and due to faulty development of the lens.

The cause of the *acquired* form is a central ulceration of the cornea, with perforation and loss of the aqueous humor, whereby the anterior capsule comes into contact with her cornea and produces opaque tissue under the capsule.

When the cataract is so large that it causes a noticeable projection of the anterior pole of the lens, it is known as a *pyramidal cataract.*

These cataracts can be seen by oblique illumination. On examination with the ophthalmoscope, it is noticed that there is no parallactic displacement, which shows that the cataract is near the plane of the pupil.

Treatment is usually not required. When of very large size an iridectomy is indicated.

A **zonular, perinuclear, or lamellar cataract** is due to opacification of the layers of the lens between the cortex and nucleus. Only a few lamellæ may be involved, in which case spiculæ, or spokes, are seen; or there may be a diffuse opacification which appears like a ring. It may be congenital or may form in early infancy. It is usually bilateral and stationary.

These cataracts are often observed in individuals who show evidences of rhachitis.

Treatment is usually not required. When vision is considerably interfered with, an iridectomy is indicated, if the periphery of the lens be normally transparent; otherwise an extraction or discission of the lens, according to the age of the patient, is necessary.

When opacities occur in the anterior or posterior layers of the cortex the condition is known as **anterior** or **posterior cortical cataract.**

The *posterior* form is more common than the *anterior,* but both may be present at one time.

These cataracts are usually due to disturbance of nutrition, as a result of disease of the vitreous humor, choroid, or retina. They remain stationary for years, but finally a total opacity occurs. Because of the damaged condition of other parts of the eye, the vision is bad, even after operation, which should not be too readily undertaken.

The **senile cataract** is the most common of all forms, and usually begins to develop after the fiftieth year. It commences as a partial opacity, which increases in size until the entire lens is involved. Both eyes are affected, one a variable time after the other.

A senile cataract is *due* simply to the degenerative changes incident to old age, and not to morbid conditions.

The *development* of a cataract from its incipiency to maturity or complete opacification requires a variable period of time—from a day to twenty years.

The senile cataract, being progressive, is divided into four stages, each of which has its characteristics and significance.

The *first stage*, or that of incipiency, presents upon examination spiculæ, or other form of opacity, which involve only a portion of the lens. The anterior chamber is of normal depth.

The *second stage*, or that of intumescence or swelling, is characterized by an increase in volume of the lens, whereby the anterior chamber becomes more shallow than normal. When a candle is held to one or the other side of the eye, the iris throws a shadow through a part of the lens, indicating that this part of the lens is transparent. The lens-star is plainly visible, the lens has a bluish-white color, and its surface appears smooth, glossy, and silky.

The *third stage*, or that of maturity, is characterized by the normal depth of the anterior chamber, due to the loss of water which in the second stage caused the intumescence. The lens is completely opaque, in consequence of which the iris no longer throws a shadow. The lens is of a dull-gray or brown color, and the lens-star can still be discerned. This is the stage for operative interference, since the lens-substance can be removed *en masse* from the capsule.

The *fourth stage*, or that of hypermaturity, presents an abnormally deep anterior chamber, the iris throws no shadow, and the lens-substance has liquefied.

Senile cataracts are hard until they become hypermature or overripe, when they are soft.

When the nuclear portion of the lens remains hard and the rest is liquid, a so-called **Morgagnian cataract** results.

An overripe cataract is subject to change from the deposition of cholesterin or calcareous material, or the anterior capsule may become thickened, forming a so-called **capsulolenticular cataract.**

Progressive cataracts are often due to the poor nutrition incident to general diseases, as diabetes. The cataracts are bilateral and usually develop with great rapidity.

Secondary cataracts are those which are the result of some ocular affection. The cataract receives its nutrition largely from the ciliary body, and hence any disease affecting its secretory function will interfere with the nutrition of the lens. Glaucoma, retinitis pigmentosa, myopia of high degree, iridochoroiditis, detachment of the retina, and iridocyclitis may lead to opacification of the lens.

A *secondary capsular cataract*, or membranous cataract, is the result of traumatism or operation when parts of the lens-substance and capsule remain in the eye and begin to proliferate. The sight may be much reduced in spite of endeavors to keep the pupil clear.

Cataract nigra, or black cataract, gives the pupil the normal dark appearance, but ophthalmoscopic examination shows that the lens is not transparent. It may be due to infiltration of blood, or to a condition of the lens in which the cortical is changed into nuclear tissue, which undergoes degenerative changes.

THE VITREOUS HUMOR.

Anatomy.

The **vitreous humor**, or hyaloid body, is a gelatinous, transparent substance which fills the space bounded by the lens in front, and the retina at the sides and behind. It represents four-fifths of the space of the eyeball, and is inclosed in a thin, transparent hyaloid membrane, which is, however, wanting at the lenticular fossa in which the posterior capsule of the lens rests.

The **hyaloid membrane** is attached to the retina (to the anterior part much more firmly than to the posterior), the ciliary body, the zonula, and most firmly to the optic disk, in consequence of the entrance of the hyaloid artery during fœtal life at this point.

The **canal** which extends from the papilla to the posterior pole of the lens is known as Cloquet's (or Stilling's) canal. In fœtal life the hyaloid artery courses through this canal, which after birth is probably a lymph-canal, the artery having disappeared. The hyaloid artery should present, if anything, only a trace after birth.

Uses: The vitreous prevents detachment of the retina and dislocation of the lens, and maintains the shape of the eye.

The vitreous has no bloodvessels, and receives its **nourishment** from the uvea, more particularly from the ciliary body.

Congenital Anomalies.

In fœtal life the vitreous contains many cells, and is therefore quite opaque. At times some of these cells remain, producing **slight opacities,** which give, however, very little trouble.

A **persistence of the hyaloid artery** sometimes occurs, and is recognized as a gray cord extending from the papilla a variable distance into the vitreous, sometimes even to the posterior capsule of the lens. The canal of Cloquet is visible when the walls are for some reason opaque.

Posterior polar and cortical **cataracts** occasionally complicate these conditions.

Injuries.

Foreign bodies projected into the vitreous or a lens dislocated from injury usually cause considerable inflammation : at times an iridocyclitis, purulent hyalitis, or panophthalmitis. Foreign bodies should be removed as soon as possible. A magnet may be employed to remove pieces of iron with some chance of success. Failing to remove the foreign body, an enucleation must be done to prevent sympathetic inflammation.

Hemorrhages into the vitreous produced by injury cause more or less opacity. If the hemorrhage be not too great, absorption may take place. The iodides and diaphoretics are possibly of some service.

Diseases of the Vitreous.

Opacities of the vitreous are the result of exudates due to inflammations of the uvea or retina or spontaneous hemorrhages. The patient notices floating particles (muscæ volitantes), visual acuity being diminished in proportion to the number of the opacities.

The *prognosis* depends upon the duration of the exudates.

Recent exudates are often amenable to *treatment;* old ones are not. The iodides and eliminants generally are useful in some cases.

Sometimes the **exudate** actually fills the vitreous chamber. A *plastic exudate* may become organized and result in atrophy of the bulb. A *suppurative exudate* usually terminates in rupture of the sclera, with loss of some of the contents of the eye—phthisis bulbi.

A *purulent hyalitis* is a purulent infiltration of the vitreous, but cannot be recognized by a yellow reflex seen through the pupil except in mild cases. The condition terminates in panophthalmitis and phthisis bulbi. *Treatment* is useless.

Synchisis of the vitreous is a liquefaction of more or less of the vitreous. This condition *complicates* diseases of the retina and choroid. It may lead to atrophy of the bulb from a diminution of the quantity of the vitreous; detachment of the retina; and softening of the zonula, which may later permit spontaneous luxation of the lens.

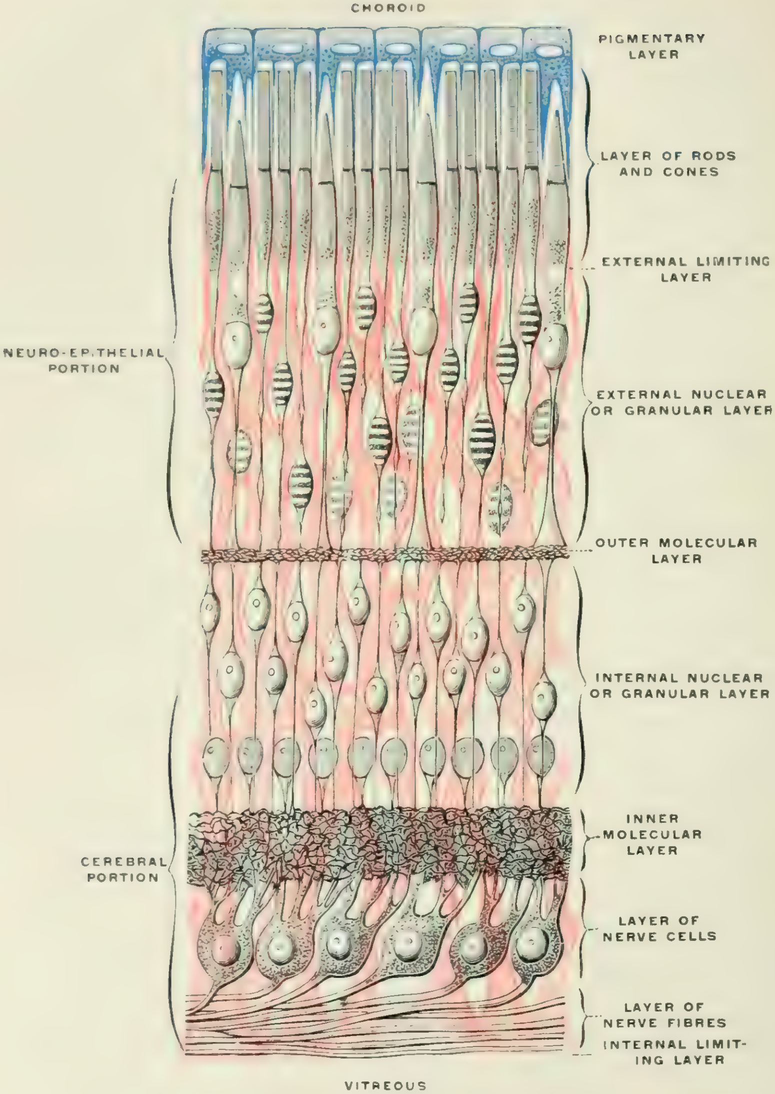
Parasites: Cysticercus cellulosæ and filaria are very rarely found in the vitreous. They should be removed, since shrinking and loss of the eye are the inevitable result. If removal be impossible, enucleation is indicated.

THE RETINA.

Anatomy.

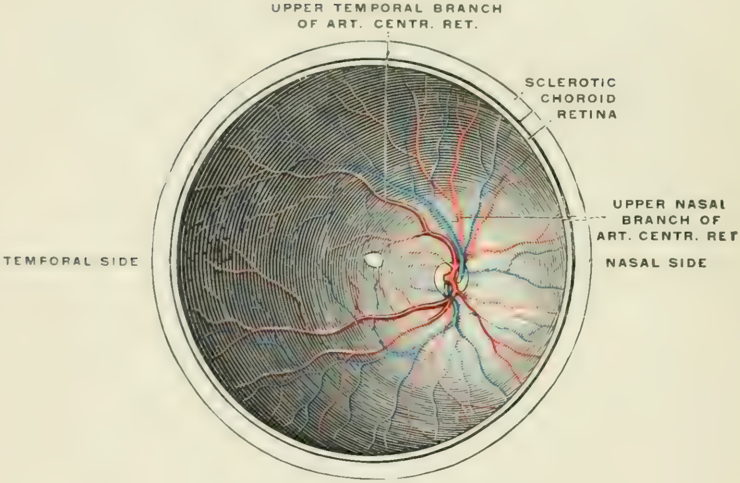
The **retina** is the inner of the three coats of the eye. It lies between the vitreous and the choroid. Because it is more firmly adherent to the vitreous than to the choroid, detachment of the retina from the choroid may occur in consequence of a loss of vitreous. The retina extends from the entrance of the optic nerve to the posterior end of the ciliary body, ending, with the exception of the tapetum, in a wavy line, the *ora serrata.*

PLATE V.



Section of Retina, showing its layers. The Fibres of Müller are in red. Diagrammatic. (Schultze.)

PLATE VI.


UPPER TEMPORAL BRANCH OF ART. CENTR. RET.
SCLEROTIC
CHOROID
RETINA
UPPER NASAL BRANCH OF ART. CENTR. RET
TEMPORAL SIDE
NASAL SIDE

Retina of the Right Eye, front view.

The Macula Lutea is seen in the middle, and the porus opticus at the right of it. (Testut.)

The part of the retina between these points is known as the *pars optica retinæ*, and is of the greatest importance. The *tapetum*, or pigment-layer of the retina, forms a single layer of epithelial cells on the inner surface of the ciliary body, and this part is known as the *pars ciliaris retinæ.* From the ciliary body this layer passes to the iris, lining its entire posterior surface. This part is known as the *pars iridica retinæ.*

The retina is quite transparent: it is thickest near the disk, where it measures $\frac{1}{75}$ of an inch, and thinnest near the ora serrata, where it is $\frac{1}{200}$ of an inch in thickness.

The retina is **composed** of ten layers (Plate V.) from within outward, as follows: (1) the internal limiting membrane; (2) layer of optic nerve-fibres; (3) layer of ganglion-cells; (4) internal plexiform, or molecular, layer; (5) internal nuclear, or layer of bipolar cells; (6) external plexiform, or molecular, layer; (7) external granular, or layer of bodies of visual cells; (8) external lining membrane; (9) layer of rods and cones; and (10) the pigment-layer. The internal and external limiting membranes and the radiating fibres of Müller constitute the support for the nervous part of the retina.

The **internal limiting membrane** is a kind of connective tissue formed by the cone-shaped extremities of the supporting fibres of Müller.

The **external limiting membrane** represents the external extremities of Müller's fibres.

The **macula lutea** is an oval spot situated one-eighth of an inch to the outer side of the papilla. At its central point is a slight depression, known as the *fovea centralis*, which is the most sensitive part of the retina. The macula represents the centre of direct vision, and the papilla, which is a little to the nasal side, has fibres which are insensible to light, and hence in the field of vision there is a corresponding blind area, known as the "*blind spot* of Mariotte" (Plate VI.).

The **arteria centralis retinæ** supplies the inner layers of the retina with blood. This vessel divides into four branches, called the superior and inferior nasal and the superior and inferior temporal. These arteries are accompanied by a corresponding vein, and do not anastomose, thereby causing much trouble when for any reason an artery becomes plugged.

The *fovea centralis* and the external layers of the retina receive their nutrition from the capillary layer of the choroid, which provides nutritive lymph.

In **normal eyes** a light streak is seen on the retinal vessels. This is supposed to be due to refraction of rays of light in passing through the bloodvessel, the posterior wall of which and underlying tissue reflecting the rays back to the observer.

Anomaly and Neoplasm of the Retina.

Opaque nerve-fibres are not, as is generally described, a congenital anomaly, since fibres in any case at birth have no medullary sheaths for some distance behind the lamina cribrosa. In the case of opaque nerve-fibres, the medullary sheaths continue their course beyond the lamina cribrosa into the bulb, and in consequence one sees on ophthalmoscopic examination white spots starting usually at the margin of the disk and ending in a brush-like extremity. The interference with vision depends upon the size and position of the patches. The blind spot is increased in size.

There is no *treatment.*

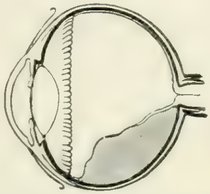
The only **neoplasm** of the retina is *glioma ;* and this occurs only in children. One or both eyes may be involved, but usually the trouble is unilateral. This neoplasm is similar to a sarcoma of the choroid in its development, and accordingly four stages are described. In the *first stage,* the pupil presents a peculiar reflex and the eye is sightless, but there is no external sign of inflammation. In the *second stage,* the eye becomes glaucomatous, with the attending pain and irritation. In the *third stage,* the growth perforates the bulb. In the *fourth,* metastatic invasions, more particularly in the liver, occur.

An early enucleation offers the only chance for life. Exenteration of the orbit is sometimes indicated, but is too often useless.

Injuries.

Traumatic detachment of the retina (Fig. 19) may be *produced* by concussion, with or without opening of the bulb, and by foreign bodies. Hemorrhages from the choroidal vessels may push the retina away from the choroid, since the retina is firmly attached only to the external tissues at the disk and ora serrata. The pressure of the vitreous under normal circumstances keeps the retina in contact with the choroid, and any considerable loss of vitreous diminishing the pressure permits a retinal detachment.

FIG. 19.



Section of eye with partial detachment of retina (Nettleship.)

Rupture of the retina sometimes occurs as a result of a diminished amount of vitreous, which in shrinking tears the retina, to which it is attached. The vitreous may then pass through the rupture, and thus separate the retina from the choroid. Rupture of the retina seldom occurs from concussion.

The **diagnosis** of *detachment* is only difficult when the vessels cannot be recognized upon the veil-like retina. A difference of refraction of different parts of the retina is demonstrable.

Rupture of the retina can be diagnosed by the interrupted course of the vessels.

The **prognosis** of traumatic detachments is not hopeless. In eyes previously sound, the retina may become reattached and the vision may not suffer to any great extent.

Treatment: As soon after the injury as possible the eyes should be bandaged, and the patient should remain on his back for some weeks.

Concussion of the retina is due to blows upon the bulb. The retina has a milky appearance for a day or two, after which, if anything, only a few hemorrhagic spots are left.

Foreign bodies sometimes remain fastened in the retina. They are first covered by blood, and finally become imbedded in a connective-tissue capsule.

They should be removed.

Traumatism may cause **rupture of the retinal vessels**, and some retinal hemorrhages have their origin in this way.

Pathologic detachment of the retina may be *caused* by spontaneous hemorrhages from the retina, retinitis (more particularly albuminuric), myopia with posterior staphyloma, intraocular tumors (especially sarcoma of the choroid), cysticercus subretinalis, orbital abscesses and tumors, or chronic inflammations of the choroid.

The *prognosis* is in general bad, but is modified by the condition of the choroid and vitreous.

Some favorable cases do well on diaphoretics and absolute rest in the recumbent position. When detachment becomes complete, enucleation is necessary to relieve pain and prevent sympathetic inflammation.

Disturbances of Blood-supply.

Hyperæmia of the retina may be active or passive.

Active hyperæmia is observed in the initial stages of inflammation of the optic nerve or retina. Meningitis, syphilis, tumors of the orbit or optic nerve, sunstroke, and a high degree of hypermetropia may produce active hyperæmia. The veins are seen to be enlarged and tortuous, and the reflexes are interrupted, since successive parts of the vein do not lie in the same plane ; the arteries remain unchanged.

Passive hyperæmia accompanies glaucoma, papillitis, orbital tumors, and general anæmia.

Anæmia of the retina may be due to loss of blood, general anæmia, an embolus of the retinal vessels, and retrobulbar neuritis. Quinine or bromine amaurosis is attended with anæmia of the papilla and retina.

Hemorrhages of the retina may take place in either the external or the internal layers. When in the *external layers*, the hemorrhages are round or irregular in form ; when in the *internal layer* of nerve-fibres, they appear in streaks, since the blood follows the course of the fibres.

Hemorrhages may be *due* to traumatism, atheromatous degeneration of the bloodvessels, extreme anæmia, active and passive hyperæmia of the retina, embolus of the central artery, thrombosis of the central vein or of its branches, or local disease of the retinal and the adjoining choroidal vessels.

Retinal hemorrhages are absorbed in a few months, and the existence of a scotoma depends upon the extent of damage done to the retinal structure.

A **total embolus** of the central artery produces marked anæmia of the retina, the arteries being reduced so much in size that they appear like thin threads. About the papilla they may be easily seen, but at a variable distance from it they may hardly be recognized. The veins are somewhat diminished in size, and the papilla is quite white and its margin is indistinct. The nutrition of the retina being interfered with, it becomes opaque to the extent of surface containing the external plexiform layer. Because this is wanting in the fovea centralis the normal red color of the fundus is here preserved.

Sudden blindness is the history given in cases of total embolism.

In a few recent cases the embolus may advance into a small branch of the central artery and a part of the retina regain its function. Generally the sight is permanently lost, since atrophy of the retina and papilla soon follows the other condition.

When the embolus is infected a suppurative retinitis and finally a panophthalmitis result.

An embolus lodging in a branch of the central artery produces a disturbance of vision corresponding with the area involved. The retinal changes occur only in that part supplied by the vessel affected.

Thrombosis of the central vein when complete causes great disturbance of vision, which soon becomes total loss of sight. The veins are tortuous and enlarged, the arteries hardly visible, the papilla is generally congested, and retinal hemorrhages are seen in all parts of the fundus. Thrombosis of the smaller branches may occur, and this produces the changes already mentioned limited to the affected part. An orbital cellulitis (more particularly when caused by facial erysipelas), valvular disease of the heart, and atheromatous degeneration of the bloodvessels may cause thrombosis.

Inflammations of the Retina.

According to the *seat* of the inflammation, whether it be in the outer or the inner layers of the retina, we distinguish **retinitis externa or interna.**

A *retinitis externa* is *caused* by general diseases, such as syphilis, which so frequently involves the choroid; and local diseases of the eye, such as myopia, which produces choroidal changes.

A *retinitis interna* is *caused* by diseases which produce degenerative changes of the vessels or a change in the composition of the blood. A *hemorrhagic retinitis* is due to the former cause; an *albuminuric, leukæmic*, or *diabetic retinitis* to the latter.

A *retinitis externa produces changes* of the pigment-layer, appearing as whitish atrophic spots, which, if the choroid be also atrophied, permit a view of the sclera. Accumulation of pigment frequently occurs at the borders of these atrophic spots. A retinitis externa is nearly always *associated with* a choroiditis, which is generally the primary disease.

A *retinitis interna is recognized* by a hyperæmia and cloudiness. The papilla is indistinct, sometimes swollen; extravasations and whitish masses occur in the supporting fibres and in the optic nerve-fibres; and the walls of the retinal vessels may suffer a change.

Retinitis syphilitica is the commonest form of retinitis, and is usually associated with disease of the choroid and iris. The disease may be circumscribed or diffuse.

When *circumscribed*, the exudate is seen near a large retinal vessel or in the macular region. The absorption of the exudate produces scars, which may by contraction cause a detachment of the retina.

In the *diffuse* form, the retina is opaque, more particularly near the larger vessels. Lymph-corpuscles infiltrate the inner retinal layers and the supporting fibres become thickened. Bluish-white masses, often larger in size than the papilla, occur, especially in the macular region, and the retina is much thickened in the affected parts. The inflammatory products may become absorbed, leaving spots under which the choroidal vessels are seen and around which is a deposition of pigment.

Atrophy of the retina sometimes occurs, and this is diagnosed by the small size of the vessels and the change of the color and indistinctness of the papilla.

The *choroid* and the *vitreous* are very prone to become involved in a syphilitic retinitis. Small dust-like particles seen in the vitreous are due to implication of the vitreous.

Antisyphilitic **treatment** should be instituted, although much cannot be promised.

Retinitis albuminurica is nearly always a bilateral disease characterized by haziness of the retina, indistinct margin of the papilla, hemorrhages, and white spots occurring about either the papilla or the macula. The white spots frequently present a stellate arrangement about the macula, and thus make this form of retinal disease characteristic.

The disease is *caused* by changes in the vessels induced by the condition of the blood incident to renal disease.

The *prognosis* of retinitis albuminurica is grave, except when caused by scarlatinal nephritis or parturition.

Retinal detachment occasionally occurs when the disease is severe. The detached part is opaque and fixed, and is globular in shape.

Retinitis leukæmica is characterized by pallor of the fundus, retinal hemorrhages, and white patches due to the exudation of white and red blood-corpuscles. Sometimes the veins appear red and the arteries yellow. White patches with red borders occurring near the equator and in the macular region are of diagnostic value in connection with pallor.

Retinitis diabetica can be *differentiated* from retinitis albuminurica by the presence of groups of small semicircular or serrated bright spots occurring between the superior and the inferior temporal branches of the central artery, by the absence of the stellate arrangement of spots about the macula, by limited opacity of the retina, and by a normal papilla.

Retinitis hæmorrhagica is the result of changes occurring in the retinal vessels. Heart disease, gout, atheromatous or calcareous degeneration of vessels in general, pernicious anæmia, syphilis, and sepsis may lead to retinal hemorrhages.

These hemorrhages often terminate in atrophy of the retina and papilla.

Sclerosis of the retina, so-called *retinitis pigmentosa*, is a bilateral disease, sometimes congenital, frequently hereditary, characterized by a slow and progressive fibrous tissue-degeneration with or without a deposition of pigment. The papilla is rather yellow, its margin is very distinct, the retinal vessels are much reduced in size, and the retina itself is no longer transparent, but appears like a veil over the choroidal vessels. *Pigmentation* at first occurs in the periphery of the fundus, and progressively invades the more central parts. The pigment-spots appear not unlike bone-corpuscles, occurring more particularly along the sides of the vessels.

The patient has frequently malformations not only of the eyes, but also of other parts of the body ; and since a third of the cases are the offspring of parents who are blood-relations, *consanguinity* would seem to be an important *cause* of the disease.

Night-blindness (hemeralopia) is the most important symptom. A concentric contraction of the visual field, but with fairly normal central vision, occurs in a typical case. When the vision is poor from the beginning, nystagmus, which renders examination difficult, is often present. A posterior cortical cataract is a frequent complication ; it may appear as a dot, a disk, or a star.

Treatment is of no avail. It is impossible to check or improve the condition, and the only comfort that can be given is the fact that the disease advances slowly. The patient should, however, be instructed to protect and rest the eyes.

Diagnosis: Retinitis pigmentosa might be confounded with a *choroiditis* or a *retinochoroiditis syphilitica.*

The pigmentary spots in choroiditis are traversed by the retinal vessels, which in retinitis pigmentosa are hidden because the deposit occurs in the inner layer of the retina. Choroiditis is associated with atrophic changes which do not occur in retinitis pigmentosa. In retinochoroiditis, the outline of the choroidal vessels, apparent through the atrophied retina, and the pigment-spots are to be seen as in retinitis pigmentosa ; yet opacities in the vitreous and choroidal changes in the periphery and in the macular region are differentiating points. In some cases the history is the only means of diagnosis.

THE OPTIC NERVE.

Anatomy.

The **nerve-fibres** which form the retina collect at a place a short distance to the nasal side of the posterior pole of the bulb, constituting the papilla, or optic disk, which can usually be seen with the ophthalmoscope, and is therefore an invaluable landmark in making ophthalmoscopic examinations.

The **disk** is a round or oval whitish spot that has a diameter of about 1.5 millimetres ($\frac{1}{18}$ of an inch). It appears nearly sixteen times larger upon direct, and four times larger upon indirect, ophthalmoscopic examination. The papilla is often surrounded by a whitish circle, known as the *scleral ring*, which represents the exposed edge of the sclera. A blackish circle exterior to this, known as the *choroidal ring*, is due to the view of the pigment which is sometimes present.

The nerve-fibres coming from the constricted head of the optic nerve diverge in all directions in the formation of the retina, causing an **excavation** of the disk. This is but exceptionally in the centre of the disk, and can be recognized by its exceeding whiteness. This excavation is physiological and is not to be confounded with pathological excavations, which may be either glaucomatous or atrophic. The **glaucomatous excavation**, or cupping, differs from the **atrophic** in that it is deeper and the lamina cribrosa is pushed back. In **physiological excavations**, a part of the disk is involved, whereas pathological excavations concern the entire disk.

The **intraocular portion** of the optic nerve is the papilla or disk, and extends back to the lamina cribrosa, which consists of the inner layer of the sclera and the modified choroid. The **lamina cribrosa** presents many perforations for the passage of the nerve-fibres, which up to this point only have sheaths. The weakest and most yielding portion of the tunics is the lamina cribrosa, and accordingly an increase of intraocular tension is first manifest by bulging at this place. Moreover, the sclera surrounding the intraocular portion of the nerve is unyielding, so that swelling of the optic nerve causes a constriction interfering with the nutrition of the retina, of which the internal layers are supplied by the arteria centralis retinæ.

The **central artery** and **vein** pass through the middle of the intraocular portion of the optic nerve, and pathological changes of this part cause changes of circulation and nutrition which may impair the sight.

The **orbital portion** of the optic nerve extends from the lamina cribrosa to the optic foramen. Its length exceeds the distance from the optic foramen to the eyeball, so that the movements of the eye are not interfered with.

The *sheaths* of this part of the optic nerve are three, corresponding with the pia, arachnoid, and dura mater. The space between the arachnoid and dura is the subdural space; that between the arachnoid and pia the subarachnoid space. These spaces communicate posteriorly with similar spaces between the meninges of the brain, and end anteriorly in blind extremities in the sclera.

The **intracranial portion** of the optic nerve extends from the optic foramen to the chiasma, and is about one-third of an inch in length. It has but the pial sheath, the others covering the nerve at its exit from the optic foramen.

The optic nerves unite in the **chiasma** in such a way that about three-fifths of the nerve-fibres of each nerve cross to the other side. Accordingly, only two-fifths remain on the same side. The decussating fibres supply the nasal half of the eye, the others the temporal half.

The optic nerve-fibres from the chiasma to their origin in the brain form the **optic tracts.** Some nerve-fibres have been traced to the cortical substance of the occipital lobe, others to the floor of the fourth ventricle to the nuclei of the origin of the motor oculi. From the intimate relation of the motor oculi to the optic nerve-fibres, it is easy to understand why light should cause a reflex contraction of the pupil, and why both pupils contract consensually, some optic nerve fibres of a single eye passing to the nuclear centres of both of the third nerves.

Malformations and Neoplasms.

Congenital malformations of the optic nerve:

A *coloboma* of the sheath of the optic nerve may be partial or complete. The cleft upon which the existence of a coloboma depends is in the under surface of the nerve, and accordingly a partial coloboma occurs at this place.

Congenital atrophy of the optic nerve produces the same picture as postnatal atrophy.

Congenital glaucoma always produces deep cupping of the disk, which can be easily demonstrated with the ophthalmoscope.

The *papilla* is sometimes apparently *reversed*, the temporal pole being turned toward the nose.

Endotheliomas, various kinds of **sarcoma, gliomas, fibromas, neuro-fibromas,** *tubercle*, and *gummata* have occurred in the optic nerve, and may not produce characteristic appearances.

Injuries.

The optic nerve may be injured by **foreign bodies**, as bullets; or **cuts** which involve the orbital structures.

Hyaline concretions are rarely found in the tissues of the papilla.

Injuries of the skull, either concussion or fracture, may damage the optic nerve, which after a time becomes atrophic.

Unfortunately little or nothing can be done in cases of optic-nerve injury.

INFLAMMATIONS.

Inflammations of the optic nerve are classified as *intraocular*, when the papilla shows evidences of disease; and as *retrobulbar*, when the disease is confined to the orbital portion of the nerve.

Papillitis.

Definition: A *papillitis*, or inflammation of the intraocular portion of the optic nerve, is a chronic disease characterized subjectively by disturbance of vision and objectively by intraocular changes.

Ophthalmoscopic picture: The disk is changed in color, its margin is indistinct, and it appears enlarged. The arteries are smaller, and the veins larger and tortuous because of their compression by the swollen nerve-head between the unyielding sclera. The papilla is actually swollen, so that it extends farther forward than usual.

When the papilla does not show signs of great inflammation and the circumpapillary portion of the retina is involved, the condition is called a *papilloretinitis.* In this disease the ophthalmoscopic picture is that of a mild papillitis with exudates in the circumpapillary parts of the retina ; the vessels may be but slightly changed in appearance.

Etiology : A papillitis may be due to intracranial, orbital, systemic, or localized diseases ; and to injuries or primary affection of the nerve.

The *intracranial causes* of papillitis are basilar meningitis, either traumatic, tubercular, syphilitic, pyæmic (especially from disease of the middle ear), or epidemic cerebrospinal meningitis ; cerebral (extrameningeal and intrameningeal) abscesses ; tumors ; aneurysm, especially in the cavernous sinus ; hemorrhage, particularly when the meninges at the base are torn ; hydrocephalus of children ; and thrombosis of the sinuses. Papillitis when due to the above causes is usually bilateral.

Cerebral tumors and *hydrocephalus* produce an increase of intracranial pressure, and the cerebrospinal liquid finds its way between the sheaths of the optic nerves, causing an œdema of the papilla, known as choked disk. Inflammatory diseases at the base of the brain may reach the papilla by simple extension along the nerve.

The *orbital causes* are tumors, orbital cellulitis of whatever cause, and pressure by the bony walls of the optic foramen.

The *systemic diseases* which produce papillitis are the acute infectious diseases, syphilis, diabetes, anæmia, chlorosis, and lead-poisoning. Uterine affections, diseases of the other eye, chorea, or myelitis may be the cause of a papillitis. As a consequence of rheumatism or predisposition thereto, papillitis has been known to occur.

The **prognosis** of a papillitis depends upon the intensity of the inflammation, but is never good. The damage to vision varies with the degree of atrophy which follows the inflammation.

The **treatment** of papillitis consists in correcting the cause, if possible, and in rest of the eyes and the use of diaphoretics and alteratives.

Retrobulbar Neuritis.

Symptoms: This affects the orbital portion of the optic nerve, and may be either *acute* or *chronic.* The papilla does not show any characteristic change, except in the later stages, when atrophy may occur. The vision is impaired, and very often there is a central scotoma.

An **acute retrobulbar neuritis** is usually due to a cold, and is best *treated* by rest of the eyes; and in the early stages by the use of diaphoretics, as pilocarpine and the salicylates. The *prognosis* is usually good.

Chronic retrobulbar neuritis is usually due to tobacco- and alcohol-poisoning; but may occur in consequence of poisoning by stramonium, lead, sulphur, chloral, and the iodides; and occasionally complicates diabetes.

The visual field is contracted, and a central scotoma, first for green and red only and later absolute, is demonstrable upon perimetric examination. Chronic retrobulbar neuritis begins in middle age of men, who are more subject to the influences of various poisons than women.

The *treatment* consists in complete abstinence from tobacco and alcohol, or removal from the poisonous influence, and the use of strychnine, which may be used hypodermatically, increasing the dose to tolerance, or administered internally in the form of a nux vomica preparation.

The *result of the treatment* is good in recent cases; not so good when considerable atrophy has already occurred.

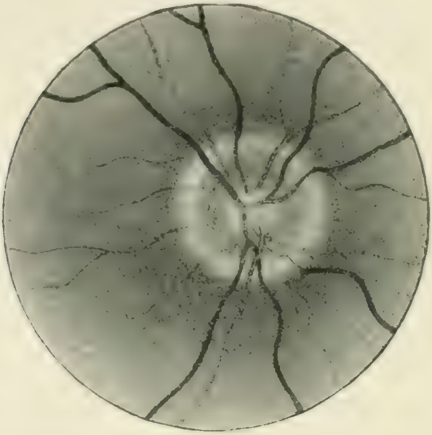
Atrophy.

Varieties: *Atrophy* (Fig. 20) of the optic nerve presents three pictures, and is accordingly classified as simple, post-papillitic, or retinitic.

Etiology: *Simple atrophy* may be due: (1) to descending degeneration of the nerve-fibres, caused by cerebral diseases which compress the optic nerve or tract; (2) to gray degeneration of the fibres in consequence of cerebral or spinal diseases;

(3) to an acute or chronic retrobulbar neuritis; (4) to an embolus of the arteria centralis retinæ; (5) to various forms of retinitis; (6) to injuries affecting the continuity of the nerve;

FIG. 20.



Ophthalmoscopic appearance of disk in atrophy following hemorrhage. (Jager.)

and (7) to exposure to cold, insufficient food, excesses, or mental emotion.

A *post-papillitic atrophy* follows a papillitis, and is accordingly due to the cause of the papillitis.

Retinitic atrophy is likewise due to the causes of retinitis, retinitis pigmentosa, and choroidoretinitis specifica, with which it frequently occurs.

The **atrophic excavation** which occurs in *simple atrophy* permits a view of the lamina cribrosa which gives the disk the stippled appearance. The lamina cribrosa is not pushed back in the atrophic excavation as it is in glaucomatous cupping. The papilla being swollen during a *papillitis*, never atrophies to the extent of producing an excavation, and this is the reason the physiologic cup is filled in.

The **prognosis** of atrophy of the optic nerve is unfavorable, yet post-papillitic or retinitic atrophy may not become progressive.

The **appearance of the fundus** of the three forms of atrophy can be understood from the following scheme:

	Simple atrophy.	*Post-papillitic atrophy.*	*Retinitic atrophy.*
Color of disk.	Bluish or greenish-white and stippled.	Hazy, bluish or grayish-white and uniform.	Dirty yellow or dark reddish-gray.
Margin of disk.	Very distinct and regular.	Not more distinct than normal, blurred in places; often irregular.	Blurred.
Surface of disk.	Slightly concave: physiologic cup not filled in.	Flat; physiologic excavation filled in.	At times concave.
Larger retinal vessels.	Normal or nearly normal in size.	Diminished in size, the arteries in particular; white lines, due to thickening of their walls, are seen along the vessels in places.	Very considerably diminished in size or absent.

The **treatment** should be instituted according to the cause. Strychnine, iodides, and mercury may be tried, but with little prospect of success.

FUNCTIONAL DISEASES OF THE EYE.

Amblyopia is a term which expresses defective vision not due to refractive errors. It may be congenital; or occur from disuse of the eye.

Congenital amblyopia may complicate various congenital anomalies of the eye, as high degrees of astigmatism or hypermetropia, coloboma of the iris, choroid or ciliary body, hydrophthalmos, and microphthalmos. It is usually unilateral, and may not be noticed for many years.

Amblyopia ex anopsia implies defective vision from disuse of the eye.

The disuse *may be due* to opacities of the cornea or lens which were caused in infancy. After development of the eye, which is completed in adult life, disuse of the organ is not likely to produce amblyopia. Strabismus occurring during infancy often leads to amblyopia which may affect half or all of the retina.

In some cases of *convergent strabismus*, the objects in the temporal half of the field of the squinting eye are seen; whereas those in the nasal half are not, from regional exclusion, which favors single vision.

The *treatment* for amblyopia ex anopsia consists in use of the eye, either by bandaging the other or by performing an operation to remove a cataractous lens. Discission may be done in infancy after three months of life, and the sooner it is done the better will be the result.

Hysterical amblyopia is frequently observed in young hysterical female subjects, and is characterized by an absence of intraocular changes together with impairment of vision. *Perimetric examination* shows a concentric contraction of the visual field, which becomes smaller the longer the patient is examined. The *prognosis* is good in most cases. The *treatment* consists in the use of tonics, electricity, and suggestion, which sometimes relieve all symptoms in a short time.

Asthenopia, or inability to use the eyes, sometimes complicates neurasthenia and hysteria. Use of the eyes causes pain or fogginess of vision, which prevent the patient from using them. Refractive errors and muscular asthenopia are not present in hysterical asthenopia. Electricity and suggestion have been found useful in some cases, but often very little is accomplished by any treatment.

Amblyopia sometimes occurs in consequence of **slight cerebral changes** involving the optic tract. The more serious cerebral diseases usually produce changes of the optic nerve, as papillitis or atrophy.

Amaurosis implies absolute blindness which is not due to changes in the refractive media. It may be temporary or permanent. Uræmic poisoning may produce temporary amaurosis; cerebral lesions, permanent blindness.

Amaurosis partialis fugax is a form of temporary blindness due to some central cause. An attack often begins with headache and vomiting, and after objects have for a short time appeared blurred, vibrating, and sparkling, blindness supervenes. Vision returns soon in most cases and the sight is not damaged.

Achromatopsia, or color-blindness, may be congenital or acquired.

When *congenital* it may be partial, in which case only certain colors cannot be distinguished; or complete, when all objects appear gray. Color-blindness for *red* is the most common form of congenital color-blindness, and males are more subject to it than females.

The *detection of color-blindness* is of great importance in railway employés, who should be able to distinguish colors in order to interpret signals. In order to test the perception of colors, skeins of worsted of many shades of the primary colors are given the person to be examined, and he should be requested to put aside all skeins which to him appear blue, red, green, etc., as the case may be.

Acquired color-blindness is due to disease of the retina, optic nerves, or optic tracts, and never to disease of the media. The loss of perception of color is gradual, some colors being recognized longer than others.

Green, red, yellow, and blue fail to be recognized in the order given in disease of the optic nerve.

Hemeralopia is a symptom of many diseases, and implies that although the vision during the daytime is good, at night it is bad, or even *nil*. Peripheral opacities of the cornea or lens in bright light do not interfere with the course of the rays of light through the contracted pupil, but interfere seriously with it in the dark, when the pupil is dilated.

When the peripheral portion of the retina is diseased, the central portion is sufficient to give good vision during the daytime, the pupil being contracted; but at night vision suffers to the extent of the retinal disease.

Hemeralopia is sometimes observed in individuals whose physical condition is below par because of insufficient or improper food. It not infrequently occurs in jaundice, chronic alcoholism, or pregnancy. The *prognosis* is good, and the success of the *treatment* depends upon the restoration of health.

Nyctalopia implies good vision at night, when the pupil is dilated, and bad during the day, when the pupil is contracted. The cornea, or lens, has a central opacity, or the retina is centrally diseased when there is nyctalopia. In chronic retrobulbar neuritis nyctalopia is a prominent symptom.

THE MUSCLES OF THE EYEBALL.

Anatomy.

The eye has two kinds of muscles, extrinsic and intrinsic.

The **extrinsic** muscles are six in number, and their function is to change the direction of the eye so that objects may be brought into the field of vision. These are the superior, inferior, external, and internal recti, and the superior and inferior oblique (Fig. 21).

The **intrinsic muscles** are the ciliary and sphincter pupillæ, which are concerned respectively in accommodation and contraction of the pupil. These, like the superior, internal, and inferior recti, and the inferior oblique, are supplied by the third nerve; and, owing to the intimate relation in the floor of the fourth ventricle of the nuclei of the nerve-fibres to the ciliary muscle, the iris, and the four extrinsic muscles, convergence, accommodation, and contraction of the pupil are associated acts.

Axes, planes, and meridians: The *sagittal*, *optic*, or *anteroposterior axis* of the bulb is a line extending from the centre of the cornea to the posterior pole. The *visual axis* is a line which extends from the yellow spot to the cornea in the direction of the object looked at. The visual and the anteroposterior axes practically coincide.

The centre of motion of the bulb is a point on the anteroposterior axis one line behind its centre.

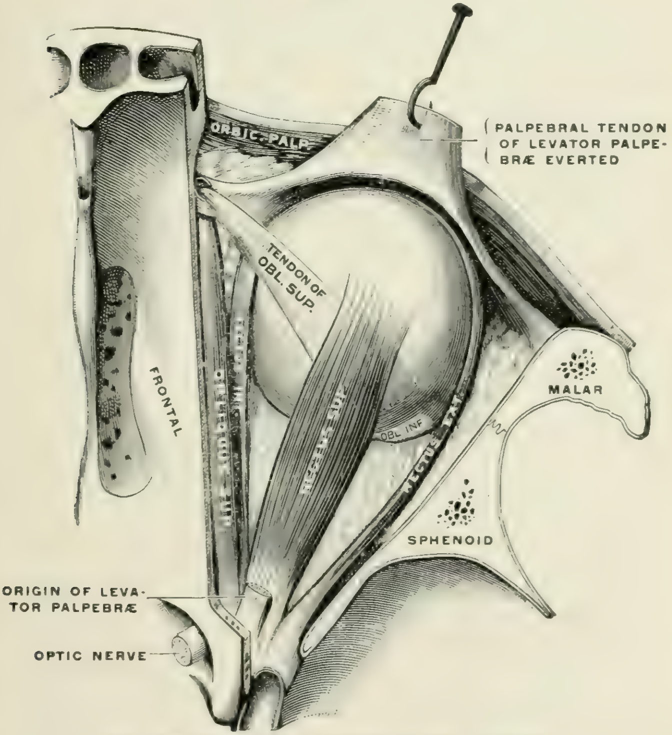
When a horizontal plane passes through the anteroposterior diameter of the bulb, it forms at its intersection with the external surface of the bulb the *horizontal meridian*.

A vertical plane passing through the anteroposterior diameter forms at its intersection with the external surface of the bulb the *vertical meridian*.

The *equatorial meridian* represents the intersection of a plane passing through the centre of motion and perpendicular to the anteroposterior axis with the external surface of the bulb.

The horizontal, or *frontal, axis* of the bulb is a line passing through the horizontal meridian at right angles to the anteroposterior axis at the centre of motion.

Fig. 21.



Muscles of right eye, viewed from above. (Testut.)

The *vertical axis* of the bulb is a line passing through the vertical meridian at right angles to the anteroposterior axis at the centre of motion.

A line connecting the centre of motion of each eye forms the *base-line.*

A plane passing through the centre of the base-line and perpendicular to it is called the *median plane.*

The *visual plane* passes through the base-line and the visual axis or lines.

The plane passed through the centres of origin and insertion of an extrinsic muscle and the centre of motion of the bulb, is called a *muscle-plane.*

Each extrinsic muscle when functioning alone moves the eye around the so-called *axis of turning*, which is perpendicular to the muscle-plane and passes through the centre of motion.

Positions of the eyeball : An eye is said to be in the *primary position* when the muscles are perfectly balanced and the visual lines are in a horizontal plane and parallel to the median plane ; other positions are *secondary.* In studying the action of a muscle the change of position of the centre of the cornea, either elevation or depression, and adduction or abduction, should be noted ; also the movement of the point of intersection of the equatorial and vertical meridians on the top of the eye.

When this point moves toward the nose it implies *nasal*, or + *wheel rotation ;* and when toward the temple, *temporal*, or — *wheel rotation.*

The **internal rectus** arises from the tendon of Zinn and the inner margin of the optic foramen, and terminates in an aponeurosis, four and one-half lines broad, which is inserted into the sclerotic coat about three lines from the internal margin of the cornea. It is the broadest of the recti ocular muscles, and pulls the cornea directly inward (adduction) and there is no wheel rotation.

The **external rectus** is the longest of the recti muscles and has two heads of origin : The upper one arises from the outer margin of the optic foramen, and the lower partly from the tendon of Zinn and partly from a pointed process of bone on the lower margin of the sphenoidal fissure. It terminates in an aponeurosis three and a half lines broad, which is inserted into the sclerotic coat about four lines from the outer margin of the cornea ; and, like the internal rectus, on a level with the centre of it.

This muscle pulls the cornea directly outward (abduction), and there is no wheel rotation since the vertical axis remains unchanged.

The **movements** of the centre of the cornea are in the line of the horizontal meridian during the action of either the *external* or the *internal rectus*, and hence there is neither elevation nor depression of the eye.

The action of either of the *other four* extrinsic muscles causes either adduction or abduction, elevation or depression, and nasal or temporal wheel rotation: their action, therefore, produces *compound motion.*

The **superior rectus** is the thinnest and narrowest of the recti muscles, and arises from the upper margin of the optic foramen and is inserted obliquely by a tendinous expansion into the sclerotic between three and four lines from the superior margin of the cornea.

The superior rectus elevates and adducts the centre of the cornea, and nasal wheel rotation takes place.

The **inferior rectus** has a common origin with the internal rectus, and is, like the superior rectus, obliquely inserted into the sclerotic, the inner edge two and a half, and the outer three and a half, lines from the lower margin of the cornea.

The inferior rectus depresses and adducts the centre of the cornea, and the wheel rotation is temporal.

The **superior oblique** arises about a line above the inner margin of the optic foramen and passes forward, upward, and inward to the upper inner angle of the orbit to a ring of fibrocartilaginous tissue, through which it continues, and is then reflected backward, outward, and downward beneath the superior rectus to be inserted by a fan-shaped aponeurosis into the sclera between the superior and external recti muscles, and midway between the margin of the cornea and the entrance of the optic nerve.

The superior oblique causes principally nasal wheel rotation, and the centre of the cornea is depressed and abducted.

The **inferior oblique** arises from a depression in the orbital plate of the superior maxillary bone, external to the orifice of the nasal duct, and, passing outward, backward, and upward, first beneath the inferior rectus and then between the external rectus and eyeball, is inserted by a fan-shaped aponeurosis into the sclerotic, near the insertion of the superior oblique. The anterior edge of the insertion is five lines from the cornea, and the posterior edge two lines from the entrance of the optic nerve.

The inferior oblique more particularly causes temporal wheel rotation, and the centre of the cornea is elevated and abducted.

Summary of Actions:

Muscle.	*Action.*
Internal rectus,	Adduction of centre of cornea.
External rectus,	Abduction of centre of cornea.
Superior rectus,	Elevation of centre of cornea. Adduction of centre of cornea. Nasal wheel rotation. Highest point of the vertical meridian moves toward the nose.
Inferior rectus,	Depression. Adduction. Temporal wheel rotation. Highest point of the vertical meridian moves toward the temple.
Superior oblique,	Depression. Abduction. Nasal wheel rotation.
Inferior oblique,	Elevation. Abduction. Temporal wheel rotation.

Superior rectus and inferior oblique—Elevation.
Inferior rectus and superior oblique—Depression.
Superior and internal recti and inferior oblique—Elevation and adduction.
Superior and external recti and inferior oblique—Elevation and abduction.
Inferior and internal recti and superior oblique—Depression and adduction.
Inferior and external recti and superior oblique—Depression and abduction.

The extrinsic ocular muscles receive their **blood-supply** from the ophthalmic artery.

The **sensory nerves** are from the trigeminus, mostly from the first division, but also from the orbital branch of the second division.

The **motor nerves** are the third, fourth, and sixth. The *third* supplies the superior, inferior, internal rectus, and inferior oblique; also the intrinsic muscles, the ciliary and sphincter pupillæ; and, finally, the levator palpebræ superioris, and explains the existence of ptosis, paralysis of accommodation, dilatation of the pupil, and divergence (paralysis of the adductors).

The *fourth*, or pathetic, nerve supplies the superior oblique; and the *sixth*, or abducens, the external rectus.

ANOMALIES OF THE MOTOR APPARATUS.

The **anomalies** of the motor apparatus of the eyes include *heterophoria; strabismus*, or *heterotropia*, which may be paralytic or non-paralytic; and *nystagmus*.

When the extrinsic, or extraocular, muscles are in perfect equilibrium and the visual lines are kept parallel with little effort, the normal condition, or **orthophoria**, exists.

When, however, a *tendency to deviation* of the eyeball occurs, and this can be overcome by special effort, **heterophoria** is the name given to the condition.

When the tendency to deviation cannot be overcome and the visual lines are not directed to the same point of regard, **strabismus, or heterotropia,** results.

Heterophoria.

Heterophorias are *divided* according to the tendency of the visual lines as follows: *esophoria*, *exophoria*, *hyperphoria*, *hyperesophoria*, and *hyperexophoria*.

Esophoria occurs when the visual lines tend to turn *inward*, owing to the superiority of action of the corresponding muscles.

To *demonstrate the presence* of an esophoria, a card should be put before one eye and the patient told to observe a fixed point with the other. On removal of the card it will be noticed that this eye makes a slight excursion outward. While the card covered the eye it was slightly turned inward. After removal of the card it is necessary for the eye to make an excursion outward in order to bring the visual lines upon the same point of regard.

The *degree* of esophoria can be determined by seating the patient at least twenty feet from a source of light (a candle answers very well) and placing horizontally before one eye a Maddox rod and before the other a prism, base out, which will bring the vertical strip or bar of light through the light.

The degree of the prism represents the degree of esophoria. It will be noted that in esophoria, the Maddox rod being before the patient's right eye, the strip of light will be to the patient's right of the light.

Convergence and accommodation must not be used in making this test to determine the muscular equilibrium, and hence a distance of at least twenty feet between the patient and light is essential to make the test of value.

Exophoria occurs when the visual lines tend to *diverge*.

When one eye is covered with a card there is a tendency to divergence; after removal of the card the eye makes an excursion inward.

With the aid of a Maddox rod horizontally placed the presence of exophoria can be determined, the strip of light being toward the nasal side of the eye before which the rod is placed.

The *degree* of the prism, with the base in, which brings the bar of light and the light in a line represents the degree of exophoria.

Hyperphoria is that condition of the ocular muscles which produces a tendency of the visual line to pass above that of the other eye. When the visual line of the right eye is above that of the left, right hyperphoria exists; when the visual line of the left eye is above that of the right, then there is left hyperphoria. The superior rectus of the right eye causes more elevation of the cornea than the superior rectus of the other in right hyperphoria. In left hyperphoria the condition is reversed.

The Maddox rod vertically placed before the right eye shows the existence of right hyperphoria when the bar of light is below the light, and left hyperphoria when above.

Hyperesophoria is the condition in which there is the compound tendency of the visual line to rise above the other and also to pass inward.

In **hyperexophoria** the visual line has a tendency to pass outward and to rise above the other.

The **causes of the heterophorias** may be *organic* or *functional.*

The **organic causes** are muscular weakness, which may be due to *smallness* of the muscle, the point of insertion, and the size of the eyeball. Weakness may also occur after exhausting diseases or previous paralysis.

The **functional causes** depend upon the association of convergence and accommodation. During the various degrees of accommodation, convergence in normal eyes is so regulated that the muscular equilibrium is perfect. Eyes with refractive errors, as hypermetropia and myopia, demand more or less accommodative effort than the normal eye. The myopia eye requires less, the hypermetropic eye more, accommodation than the normal eye, all fixing for the same distance.

In myopia, less demand being made upon the accommodation, less is required of the internal recti ; and hence a weakness, or insufficiency, of the internal recti may develop.

In hypermetropia more accommodative effort is required to see well near by than in emmetropia, and hence the demands on the internal recti are greater than are normally required, resulting in increase of function, development, and strength. The internal recti would then be relatively stronger than the external.

Insufficiency, or weakness of the interni manifests itself, when accommodative effort is prolonged for near use of the eyes by indistinct vision, diplopia, and headache. This constitutes **muscular asthenopia,** which is characteristic in that the symptoms subside when one eye is closed and no convergence is necessary.

The **treatment of the heterophorias** depends upon the amount of trouble they occasion and the cause. When no disagreeable symptoms, either ocular or reflex, occur, they may be disregarded. Refractive errors should in every case be corrected, and special attention should be given to the correction of astigmatism when it is present. Prisms may be employed in some cases when the degree of the heterophoria does not exceed 6 degrees. A prism of 3 degrees could be used before each eye to get the effect of 6 degrees, for the weight of the glass and the dispersion of light militate against the use of strong prisms.

In *esophoria* the proper prism with the base out may give relief; in *exophoria* the prism should have the base in; and in *hyperphoria* the base should be down.

It will be observed that the base is always directed to the side of the weaker muscle.

In order to develop the weaker muscles they may be exercised by the use of prisms so placed that an additional effort is imposed upon the muscle to be exercised. These prisms may be mounted in a so-called *grab-front*, and used by the patient by attaching it to his spectacles (of which most patients have need) several times a day for some minutes.

In some cases the only treatment is a *tenotomy* of the stronger opposing muscle. The tenotomy may be partial or complete according to the effect desired. The partial tenotomy, involving only a part of the tendinous expansion of the muscle, may be graduated according to the exigency of the case. The effect of such a tenotomy should be watched, and if necessary repeated until the desired result is obtained.

Low degrees of heterophoria when subject to variations should not be operated upon.

It should be kept in mind that a hyperphoria of *two degrees* may cause great distress when an esophoria or exophoria of such a degree may not be noticed, the excursions of the eye from side to side being much greater in amplitude than those up and down.

Strabismus.

Strabismus is classified as *paralytic* or *non-paralytic*.

Movements of the eye: In *non-paralytic strabismus* the movement of one eye is always accompanied with a similar movement of the other, and hence this form is called *concomitant strabismus.*

The movement of the eyeball in *paralytic strabismus* is entirely deficient to the extent of the muscle or muscles paralyzed, whereas the movement of the eyeball in concomitant squint is the same in both eyes.

Deviations: The *primary deviation* of *concomitant squinting eyes* is the difference between the position of the squinting eye when the other is fixing and the position of the same eye when it is fixing. *Secondary deviation* is the difference between the position of an eye when fixing and its position when the other eye is fixing. In concomitant squint the primary and secondary deviations are equal.

In a *paralytic squint* the *primary deviation* represents the angle between the line of vision and the line from the object to the nodal point of the paralyzed eye. If a card be placed before the sound eye, it will deviate more than the paralyzed eye when the sound eye was fixing; this is the secondary deviation. In non-paralytic squint the primary and secondary deviations are equal; in paralytic squint the secondary deviation exceeds the primary.

The **symptoms of a paralytic squint** are: loss of motility; diplopia; vertigo; compensatory position of the head; the secondary deviation is greater than the primary; and there is false projection.

The **loss of motility** depends upon the muscles paralyzed. When the third nerve is affected, all the extraocular muscles except the external rectus and superior oblique may be paralyzed. The eye protrudes slightly, since three of the recti are lax; the eye is turned outward and downward; there is ptosis; the pupil is dilated and accommodation suspended. When all the extraocular and intraocular (ciliary muscle and sphincter pupillæ) muscles are paralyzed, the condition is called *ophthalmoplegia totalis;* when the extrinsic ocular muscles are paralyzed, *ophthalmoplegia externa;* and when the intrinsic (ciliary and sphincter pupillæ) muscles are affected, *ophthalmoplegia interna.*

When paralysis affects both eyes in such a way that associated movements are not possible the condition is known as *conjugate paralysis.*

Diplopia: *Binocular diplopia* occurs in the more recent cases, and is due to the fact that the visual axis of one eye deviates from the point of regard. *Homonymous diplopia* occurs when there is undue convergence of the eyes, and the right eye sees the image to the right side and the left eye the image to the left.

When the images are crossed, the image to the left being seen by the right eye and the image to the right by the left eye, the condition is called *heteronymous* or *crossed, diplopia.* This occurs in divergence of the eyes.

Both forms occur in paralytic squint according to the kind, but evidently diplopia appears when only the eyes are moving in the direction requiring the function of the paralyzed muscle.

Vertigo and compensatory position of the head: The patient, in order to prevent vertigo and diplopia, *holds the head* in such a way that the affected muscle does not come into play. As a rule, the head is turned in a direction opposite to that of the functioning muscle.

The **secondary deviation** is greater than the primary because the extra impulse to a paralyzed muscle of the diseased eye causes but slight response; whereas the similar impulse to the corresponding muscle of the other eye produces the full effect.

False projection occurs in an eye with a paralyzed muscle, the sound eye being covered when the function of that muscle is required to see an object. The patient believes that he sees the image at a point corresponding with the impulse to the paralyzed muscle.

In *recent paralyses*, the diplopia, vertigo, and compensatory position of the head will be well marked; but in *old cases* the retina of the diseased eye learns to exclude the images, and these symptoms are not present. In old paralyses contracture of the antagonists may occur.

The **etiology** of paralyses of the ocular muscles may be due to intracranial or orbital lesions.

These are very often the result of syphilis; but cerebral diseases (particularly those at the base of the skull), tuberculosis, diabetes, diphtheria, injury, rheumatism, and exposure to cold are also causal.

The **treatment of paralytic squint** will depend upon the cause. When there is a syphilitic history, mercury and the iodides should be administered. In rheumatic paralyses the salicylates and diaphoretics are indicated. Use of the constant current is applicable to all recent cases. The paralyzed muscle may also be exercised with the aid of prisms. The employment of prisms is sufficient to relieve the diplopia and vertigo when the squint is of low degree; but an opaque plate before the paralyzed eye is necessary in bad cases. When contracture of an antagonistic muscle occurs, the condition may be remedied by a tenotomy of this muscle and an advancement of the paralyzed one. Complete paralyses are incurable.

Varieties of non-paralytic or concomitant strabismus: This is *convergent* when one eye turns inward; *divergent*, when one eye turns outward; *monolateral*, when the same eye always squints; and *alternating*, when sometimes one and sometimes the other eye squints. It may be *periodic* or *constant*.

Etiology of concomitant strabismus: It may develop from a heterophoria or be due to a visual disturbance of one eye, such as a refractive error, opacities of the cornea or lens, and intraocular disease.

Convergent strabismus is very often associated with hypermetropia. This is due to the fact that hypermetropes require a great effort to accommodate for near vision, and because of the connection between convergence and accommodation sufficient accommodation requires a strong impulse toward convergence. Hypermetropia when associated with a visual disturbance which is the result of a congenital condition or postnatal disease is likely to lead to strabismus. When the eyes are first required for near vision—that is, between the age of three and six—strabismus most frequently develops. Very often the squint is periodic, being noticed only when the child fixes for near objects; later it may become constant.

The **treatment** of *convergent strabismus* should always begin with a correction of the refractive errors. Children should not be operated upon before six years of age, when they begin to attend school and require their eyes for near work. Children under six years should be refracted after a retinoscopic examination, and the full correction should be prescribed. It is also well to keep the eyes under the influence of a cycloplegic (atropine) for several months. In order that the squinting eye may not become amblyopic from disuse, its employment should be forced by bandaging the other eye. Spontaneous cure sometimes takes place.

If after faithful trial of non-operative measures the strabismus does not diminish, a *tenotomy* of the internal rectus must be performed. When alternating strabismus is present, the eye with worse vision should be first operated, and if after one or two months the squint is still present, the internal rectus of the other eye should be tenotomized. An advancement of the external rectus together with tenotomy of the internal rectus is seldom required.

Divergent strabismus is very frequently associated with myopia. For near vision a myope needs little or no accommodation, and the impulse to the internal recti is accordingly small. As the myopic eye is often increased in size, its inward movement is also mechanically interfered with. When the convergence is insufficient for near vision one eye turns out. Divergent strabismus begins in youth (children are very seldom myopic), and tends to increase with age. An operation has only a cosmetic effect, for vision is very rarely restored. The simple operation of tenotomy of the external rectus is seldom sufficient, an advancement of the internal rectus being also necessary. In some cases nothing helps.

Nystagmus.

Nystagmus is a condition of the eyeball in which rapid, lateral, vertical, or rotatory movements take place. It usually affects both eyes when the movements of whatever kind are simultaneous.

Nystagmus may be *due* to amblyopia which developed at an early age; congenital anomalies; great refractive errors; or retinitis pigmentosa. It also occurs with cerebral diseases, and develops in coal-miners because of the position in which they are sometimes obliged to work. Coal-miners' nystagmus stops when the work is definitively discontinued. Other forms of nystagmus are not amenable to *treatment*, and the correction of coexisting refractive errors will be found unsatisfactory.

REFRACTIVE ERRORS.

Varieties.

As regards its refractive condition, the eye is either emmetropic (normal) or ametropic (abnormal). In an **emmetropic eye** parallel rays from distant objects (twenty feet and more) are focussed on the retina without any accommodative effort.

When the rays do not focus on the retina, the conditions being the same, the eye is **ametropic.**

When parallel rays passing through all meridians of the cornea focus in front of the retina, the ciliary muscle being relaxed, the condition is called **myopia.**

When the rays focus behind the retina, the condition is one of **hypermetropia.**

When the parallel rays passing through one meridian of the cornea focus on the retina and others, passing through a meridian at right angles to the first, focus in front of the retina, the condition is called **simple myopic astigmatism**; when behind the retina, **simple hyperopic astigmatism** is present.

When parallel rays passing through two meridians at right angles to each other focus at different points in front of the retina, **compound myopic astigmatism** is present.

When the rays focus at different points behind the retina the condition is one of **compound hyperopic astigmatism.**

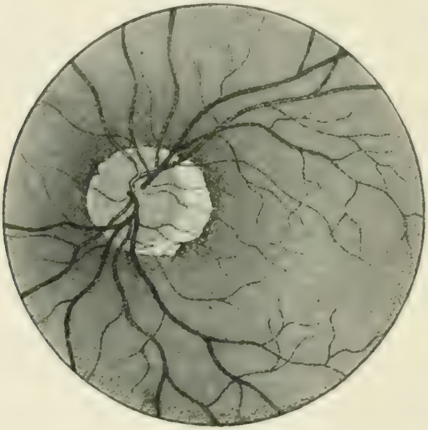
When the refraction of the two eyes is different, the condition is called **anisometropia.**

Myopia.

Etiology: Myopia may be due to an increase of the refractive power of the eye or to an elongation of its axis. An increase of corneal or lenticular curvature, an increase of density of the lens, and spasm of the ciliary muscle, whereby the convexity of the lens remains at its greatest point, are the causes of abnormally great refracting power of the eye. *Axial myopia* is due to distention of the sclera, and when the bulging occurs at the posterior part of the eye it forms the so-called *posterior staphyloma.* There may also be a displacement of the inner or the outer layer of the sclerotic, producing a change in shape of the discal end of the intervaginal space (Fig. 22).

In order **to determine the degree** of myopia, the patient should be seated twenty feet from the test-types, and concave glasses, beginning with the weaker and gradually increasing, should be put before the eye to be examined: the weakest concave glass which gives the best vision represents the degree of myopia. The degree of myopia varies from a very low amount to twenty, or rarely, thirty dioptres.

Fig. 22.



Ophthalmoscopic view of change in shape of discal end of intervaginal space. (Jager.)

In prescribing glasses for myopia of two or even three dioptres, glasses are not necessary for near work, but are usually very desirable for distant vision. When astigmatism is also present, its correction is often of advantage for near work. When the myopia is less than seven dioptres, one pair of glasses not quite correcting the myopia suffices for near and distant vision when the accommodation is good. If not good, two pairs of glasses should be prescribed, one quite correcting the myopia for distance and a weaker one for near work.

When the myopia exceeds seven dioptres, two pairs of glasses will generally be required.

Hypermetropia.

Etiology: Hypermetropia may be due to diminished refractive power of the media or to a shortening of the axis of the eye. A flattening of the cornea and diminished density or absence of the lens diminish the refractive power of the eye. Axial hypermetropia may be congenital or due to protrusion of the retina.

When a hyperope has not the necessary accommodative power to see distinctly at twenty feet, the strongest convex glass necessary to produce distinct vision represents the degree of **manifest hypermetropia.**

The convex glass necessary to give distinct vision at twenty feet when the ciliary muscle is paralyzed, represents the **total hypermetropia.**

The difference between the total and manifest hypermetropia is the amount concealed by the accommodation, and is called the **latent hypermetropia.**

When the power of accommodation of a hyperopic eye is reduced to *nil*, or is insufficient to bring parallel rays to a focus on the retina, the condition is called **absolute hypermetropia.**

The **amplitude of accommodation,** which is the difference of refractive power of the eye when at rest and when accommodation is exerted to the utmost, is the same in a hypermetropic and an emmetropic eye; but the near point of the former is at a greater distance from the eye than that of the latter. In consequence of the extra effort of accommodation necessary for near vision the ciliary muscle becomes tired, and this condition is known as **accommodative asthenopia,** not to be confounded with *muscular* and *nervous asthenopias.*

The **treatment of hypermetropia** with convex glasses is very satisfactory. A hypermetropia less than 1 D. after use of atropine can usually be disregarded. When it exceeds 1 D., as a rule, a glass should be prescribed weaker by 0.25 D. for every dioptre of hypermetropia. In children more may be deducted with advantage; and when the degree of hypermetropia in adults exceeds 3 or 4 D. the same exception to the rule applies.

Presbyopia.

Etiology: Presbyopia—"old sight"—is the result of a senile change to which every eye is subject about the age of forty years. It is due to the impaired elasticity of the lens, in consequence of which its convexity is insufficient for near work.

Treatment of presbyopia in emmetropic eyes: The aid of convex glasses supplies the want of elasticity which increases with age up to a certain point. As the inherent elasticity of the lens and its consequent convexity diminish, so the near point at which perfect vision is possible recedes from the eye. When presbyopia begins this point is 22 cm. The table below gives the distance of the near point from the eye, and the strength of the convex glass necessary to bring this point to 22 cm. in emmetropic eyes for the various ages.

PRESBYOPIA TABLE FOR EMMETROPIC EYES.

Age.	Distance of *p*.		Pr. expressed by the lens necessary to bring *p* to 22 cm. or 9″.	
	Cm.	Inches.	Dioptres.	Paris inch scale.
40	22	9	0	0
45	28	11	+1	$+\frac{1}{36}$
50	43	17	2	$\frac{1}{18}$
55	67	27	3	$\frac{1}{12}$
60	200	72	4	$\frac{1}{9}$
65	Infinity.		4.5	$\frac{1}{8}$
70	Acquired H = 1 D.		5.5	$\frac{1}{6\frac{1}{2}}$
75	" H = 1.5 D.		6	$\frac{1}{6}$
80	" H = 2.5 D.		7	$\frac{1}{5}$

The above table applies only to *emmetropic eyes;* hence it is necessary to consider the **treatment when the eyes are ametropic.** The visual acuity in every case for twenty feet should be determined, and the glass found which gives the best vision. If the patient is a *hyperope*, the glass representing the manifest hypermetropia should be added to the convex glass recommended for an emmetropic eye of an individual of the same age; if he be a *myope*, the amount should be subtracted. In every case the correction should be tried with test-types for near vision, and changes, if necessary, be made.

Astigmatism.

Varieties: *Astigmatism* is either *regular* or *irregular*. When the curvature of the refracting media is regular in every meridian, however different the curvatures of any two meridians may be, the astigmatism is regular; when the curvature in a meridian is not regular—that is, not the same throughout—the rays passing through it do not focus at a point, and the astigmatism is irregular. Regular astigmatism can be corrected by proper cylindrical glasses, but it is not possible to correct the irregular form.

Regular astigmatism may be corneal or lenticular, or both. The amount of corneal astigmatism and its axis can be quickly and objectively determined with the aid of an ophthalmometer. Retinoscopy affords a method of determining the amount of a regular astigmatism of whatever kind, and approximately the axis. Subjectively the astigmatism can be determined with the aid of cylindrical lenses and an astigmatic chart. The axis of the cylinder which is necessary to make all lines of the chart equally distinct will be at right angles to the meridian which requires correction, and the strength of the cylinder is the degree of the astigmatism. The amount of astigmatism represents the difference between the greatest and least curvatures in two meridians, called the principal meridians, which are at right angles to each other. Very frequently one meridian is vertical, the other horizontal. As a rule, the vertical meridian has the greater curvature; when, however, the horizontal has the greater curvature, the astigmatism is said to be *against the rule*.

When the refraction of one meridian is emmetropic and the other hypermetropic, the condition is one of *simple hyperopic astigmatism*; when emmetropic in one meridian and myopic in the other, *simple myopic astigmatism*. When both meridians are hypermetropic (one, of course, more than the other), the condition is called *compound hyperopic astigmatism*; when both are myopic, *compound myopic astigmatism.* When the refraction is myopic in one of the principal meridians and hypermetropic in the other, the condition is called *mixed astigmatism.* The **treatment of astigmatism** consists in the use of a cylinder, the axis of which is placed at right angles to the meridian requiring correction. Simple astigmatism under 0.5 D. does not give much trouble unless the meridian is not vertical or horizontal. It should then be corrected. The full correction of the astigmatism should always be given unless it exceeds 6 or 7 D., when a deduction is frequently necessary. For **irregular astigmatism** cylinders are useless, but the employment of a stenopæic slit is sometimes of value.

Anisometropia may be corrected with the proper glasses; but when the difference of refraction is great they will not be satisfactory.

Determination of Refractive Errors.

To determine refractive errors and properly correct them constitutes the major part of ophthalmic practice. These errors are frequently associated with disturbance of muscle-balance, and this materially complicates the work. The existence of refractive errors can be often ascertained by taking the patient's vision, each eye separately, with aid of the test-types, and in every case the examination should begin in this way. The use of a *cycloplegic* will, it is believed, give the best results in patients under forty years of age, and accordingly atropine should be used when the patient can sacrifice the time. In children atropine should always be employed; in adults, who cannot spare the time, homatropine may be used, and in many cases with good results, although the paralysis following instillation of homatropine is not equal to that after use of atropine. After relaxation of the ciliary muscle the refractive condition of the eye can very nearly be determined.

The use of the various *lenses* of the test-case after ophthalmoscopic and possibly retinoscopic examination will disclose the refractive error.

For the examination of *presbyopes* a cycloplegic is not necessary, and may be dangerous in very rare cases from increase of intraocular pressure.

In correcting the **heterophorias** it is well to ascertain the adduction, abduction, and circumduction at twenty feet. Abduction is determined by placing before a patient's eye the strongest prism, base *in*, which does not produce diplopia not to be overcome; adduction, by placing the strongest prism, base *out;* and circumduction, base *down*. The power necessary to overcome a prism, base placed *down* before the right eye, represents the right circumduction; before the left eye, left circumduction. Normally, abduction is about 7 degrees; adduction, from 30 to 40 degrees according to practice; and circumduction, 2 degrees. A case of heterophoria should not be *operated* upon until the effect of spherical, cylindrical, and prismatic glasses has been tested.

When prisms give relief after spherical and cylindrical glasses have failed, the patient may be operated upon if, for esophoria or exophoria, a prism of at most 6 degrees does not correct the trouble.

A hyperphoria of 2 degrees is often relieved by a graduated tenotomy. Heterophorias of high degree require surgical interference.

OPERATIONS.

Lid Operations.

Trichiasis may be relieved by ablation, transplantation, elevation of the bed of hair-follicles, or by correcting the position of the tarsus:

For **ablation**, the lid is supported on a horn plate, and with a small scalpel an incision about 3 mm. deep is made through the free border of the lid between the orifices of the Meibomian glands and the cilia the whole length of the lid; the flap containing the cilia is then excised.

For **transplantation**, a horizontal fold of skin is excised, and an incision as for ablation, but slightly deeper, is made. The wound of the integument is then sutured, whereby the bed of follicles is drawn up in proportion to the size of the excised skin. The excised skin is trimmed to fit the exposed surface below and grafted.

The bed of hair-follicles can be drawn up by **Hotz's method**: An incision is made across the upper margin of the tarsal cartilage of the upper lid and the exposed part of the orbicularis is excised. Three or four sutures are used to close the wound in such a way that the tarsal cartilage is included in each suture, giving the skin firm attachment to the cartilage.

The position of the tarsus may be corrected, **Snellen's operation**, by excising from it a wedge-shaped piece after the skin has been incised and the muscle excised, as is done in the Hotz operation. The lower border of the lid is everted and kept in this position by several sutures. Each suture is armed with two needles, one of which is introduced into the upper border of the tarsus to give firm support; and, after the suture has been drawn through the cartilage, both needles are pushed through the integument of the lower lip of the wound at a short distance from each other. The needles are then removed, and each suture is tied upon a bead and finally attached to the forehead with plaster.

Spasmodic entropion of the lower lid occurs in old people when there is relaxation of the superabundant skin and contraction of the orbicularis muscle. To relieve this condition, a horizontal fold of skin equal to the breadth of the lid should be excised, and also the part of the orbicularis exposed by the wound. The instruments necessary are a horn plate, scalpel, scissors, and forceps.

Organic entropion frequently causes trichiasis, and the operations suggested for the latter are frequently applicable to the former.

Ectropion of the lower lid of old people due to relaxation of the tissues may be improved by removing a V-shaped piece of integument and bringing the edges of the wound in apposition. When the ectropion is slight with little loss of tissue, a V-shaped incision involving the scar producing it is made, and the flap dissected up until the lid is no longer everted. When the lower edges of the wound are sutured, the

V becomes a Y. The operation is completed by suturing the arms of the Y.

When the ectropion is extreme and there is considerable loss of tissue, a **blepharoplasty** is necessary. To perform this, a flap may be transplanted from the neighboring skin with a pedicle; the adjacent skin may be pushed into the gap; or small dermic or epidermic grafts may be used.

A **canthoplasty** is an operation designed to lengthen the palpebral fissure at the outer canthus. The lids are separated by a speculum or with the fingers of the left hand, and the canthus cut with a pair of strong scissors. The skin, muscle, conjunctiva, and external palpebral ligament are divided by the cut. The ocular conjunctiva near the incision is dissected up and included in the sutures, of which three are necessary —one at each margin of the wound and one at the angle.

A **tarsorrhaphy** is an operation designed to shorten the palpebral fissure. This may be accomplished by excising the lid-margins of both lids, including the roots of the cilia near the outer canthus, and stitching the denuded surfaces together.

Operations for ptosis, particularly the congenital form, and any other which has not been successfully treated by other methods, have been devised by Wilder and Panas.

To perform **Wilder's operation**, general anæsthesia is necessary. It is done as follows: After the eyebrow has been shaved, an incision about one and a half inches in length is made just above the margin of the orbit and parallel with it down to the periosteum. The lower lip of the wound is then retracted, and the integument and muscle are dissected from the underlying tarso-orbital fascia until the tarsus is partially exposed. Two sutures of fine sterilized silk or catgut, each of which armed with needles at both ends, are then required. The first needle of one suture is pushed through the upper part of the tarsus about the middle of the outer half, in order to secure a firm hold for the suture, and is then passed through the tarso-orbital fascia vertically upward toward the margin of the orbit in such a way that a purse-string suture results.

Finally, the needle should include the muscle and connective tissue of the upper lip of the wound.

The second needle is introduced into the tarsal cartilage a little to the nasal side of the point where the first needle was introduced, and passed through the tarso-orbital fascia in the same manner as was the first needle, and after it has included the muscle and connective tissue of the upper lip of the wound both ends of the suture may be drawn up to the extent necessary to relieve the ptosis. The second suture is introduced in the same way as the first, about the middle of the inner half of the tarsal cartilage, and then both sutures are tied after sufficient traction has been made.

After cleansing the wound, the operation is finished by coapting the edges of the primary incision with the necessary sutures and applying a firm bandage. These sutures should be removed in five or six days, but the deep sutures allowed to remain. Removal of the deep sutures because of suppuration does not modify the success of the operation.

Operations on the Lachrymal Apparatus.

To **probe the nasal duct,** a lachrymal probe, of which various sizes should be at command, is necessary. If possible, pass a probe of middle size through the punctum of the lower lid after it has been dilated, and push it horizontally inward in the canaliculus until the bony wall is felt.

When great difficulty is experienced in passing the probe through the canaliculus, it is necessary to **slit it up.** This can be accomplished by passing the point of a canaliculus-knife vertically into the puncture, and after turning the instrument (with the cutting-edge directed upward) in a line with the canaliculus it is pushed through until the resistance of the lachrymal bone is felt. The handle of the knife is then depressed and slightly withdrawn, to divide the internal neck ; and having been again introduced to the limit of the sac, the instrument is brougnt into a vertical position, the cutting-edge of the knife being so held that the incision is slightly directed inward. This favors drainage. This operation is to be done only when absolutely necessary to permit the passage of the probe.

When the end of the probe touches the inner wall of the sac, it is turned vertically upward, and by firm and constant pressure pushed downward, slightly outward, and backward through the nasal duct to the inferior meatus of the nose. Syringing of the nasal duct is contraindicated immediately after any cutting operation of these parts.

To **divide a stricture** of the nasal duct, a strong, narrow knife may be used like a probe, and pushed through the nasal duct, care being taken not to make a false passage. The operation for cutting a stricture of the nasal duct is only a preliminary one for the passage of the lachrymal probes, which should be used long enough to establish a permanent opening.

Tenotomy and Advancement.

Tenotomy of the *internal rectus* is frequently performed; the *external rectus* is less seldom operated on; and the other muscles are rarely touched.

To do an ordinary **tenotomy** the following *instruments* are necessary: a speculum, a fixing-forceps, Stevens' straight forceps, a large and small squint-hook, and a curved strabismus-scissors.

A horizontal fold of conjunctiva is grasped by the forceps over the insertion of the muscle, and with the scissors an incision is made at right angles to the fold. This cut is then enlarged transversely across the insertion of the tendon by a snip of the scissors above and below. The conjunctiva is then pushed aside, which brings the capsule of Tenon into view. This is to be incised. Holding the wound open with the small forceps, the hook with the end downward is passed through the wound and kept in contact with the sclerotic. When the bent part of the hook is in the wound the instrument is turned around so that the end of the hook is upward. The end of the hook being next to the sclerotic when the instrument is brought upward, the hook must pass between it and the tendon. The tendon being engaged, the forceps can be dispensed with, and after transferring the hook to the left hand the scissors is passed into the wound between the sclerotic and the hook and the fibres of the tendon cut. The hook should be moved around toward the cornea, above and below, to be certain that all fibres have been divided. If the conjunctival wound is large, one suture may be employed to close it; otherwise the wound may be left alone.

The effect of a tenotomy upon the *internal rectus* is likely to increase with time, and may amount to 4 mm.; the effect on the *external rectus*, on the other hand, diminishes with time and never exceeds 2 mm.

The effect of *graduated tenotomies*, in which the tendon is button-holed or only partially cut, is still uncertain, being highly prized by some and considered useless by others.

An **advancement** is done when the effect of a tenotomy is insufficient or excessive.

To perform an advancement, general anæsthesia is usually necessary, and the instruments used for a tenotomy, with the addition of an *advancement forceps,* will be required.

The tendon is brought into view by vertical incisions through the conjunctiva and the capsule of Tenon. After the tendon has been firmly fixed in the advancement forceps it may be cut near its insertion. A silk suture is carried through the middle of the cut end of the tendon from before backward (including the conjunctiva and Tenon's capsule), and the needle, passed beneath the conjunctiva and capsule of Tenon, is brought out near the lateral edge of the cornea. Similar sutures are passed through the upper and lower parts of the extremity of the tendon; the upper one, after being carried beneath the conjunctiva and fascia, is brought out above the upper margin of the cornea; the lower suture after a similar course is brought out at the lower margin of the cornea. The advancement forceps may be now removed. The middle suture is then tightened sufficiently to produce the required effect and tied. The upper and lower sutures are then tied and the operation is finished.

The eye should be bandaged for a few days and the sutures left in place, if possible, for five or six days.

An advancement of a muscle is usually combined with a tenotomy of the antagonist.

Enucleation and Evisceration.

Enucleation is the removal of the eyeball from the capsule of Tenon with preservation of the muscles, conjunctiva, and other orbital tissues. The *indications* for an enucleation are: injuries of such kind that the eye is inevitably lost, absolute glaucoma when other treatment is useless, malignant tumors, conditions from which sympathetic inflammation may occur, ectasias not amenable to treatment, ocular hemorrhage which cannot be arrested, and a blinded and disfiguring eye. The necessary *instruments* are: a speculum, a strabismus-scissors, a heavy curved scissors, a fixation-forceps, and a large squint-hook. General anæsthesia is necessary for this operation.

The ocular conjunctiva is divided around the cornea and as near as possible to it. The capsule of Tenon is then opened and the external, superior, and inferior recti muscles and connective tissue are divided close to the bulb, with or without the use of the squint-hook. When the three recti have been divided the eye can easily be dislocated forward, and with the heavy curved scissors the optic nerve can be cut. The internal rectus and the obliques are then cut close to the bulb, and the operation is finished. Hemorrhage is not considerable, and is controlled by pressure. The conjunctival wound may be closed by sutures, but this is not necessary. An artificial eye may be worn three weeks after the operation.

An **evisceration** of the bulb consists in removal of everything within the sclera, particular care being taken that none of the choroid is left. The cornea and adjacent sclera is cut away and the contents of the bulb are removed with a spoon. The conjunctival edges are brought into apposition with sutures; but the stump is not better than that left after enucleation unless a glass ball be included in the scleral cavity. This, however, cannot be well tolerated and is often expelled.

Corneal Operations.

Paracentesis of the cornea is performed with a needle, which is thrust preferably into its lower and outer part. When the needle enters the anterior chamber the instrument should be brought into a plane parallel with the iris, to avoid wounding it.

The **corneal section** made for relief of bad ulcers is performed with a Graefe knife. This is to be plunged through sound corneal tissue into the anterior chamber, across which it is carried and pushed out of the cornea in sound tissue at the opposite side. The wound heals quickly unless it is probed too often.

Conical cornea is best tested by **removing the apex** of the cone. The knife in making the incision should enter the anterior chamber. Atropine is to be instilled and the bandage applied until the wound is healed.

Operations on Iris and Sclera.

Iridectomy is an operation whereby a portion of the iris is removed.

The **indications for such an operation** are: to make an artificial pupil; to diminish the intraocular pressure; to prevent the recurrence of iritis; to remove foreign bodies; and to facilitate the extraction of a cataract.

An *artificial pupil* is desirable, the retina and nerve being sound, when there are dense opacities of the cornea or lens which do not extend to the periphery; when the pupil is occluded; or when other treatment for a dislocated lens is useless. *Increase of tension* due to primary or secondary glaucoma is often relieved by an iridectomy, which, if made for this purpose, necessitates the removal of a large portion of the iris as near as possible to its ciliary attachment. After subsidence of inflammation following a *recurring iritis* an iridectomy at times prevents a recurrence. *Small cysts* and *tumors* of the iris and *foreign bodies* often require the removal of the segment of iris in which they are developing or imbedded. A preliminary iridectomy may be performed to facilitate an *extraction of a cataract* when there are old adhesions and other complications. It may also be done to *hasten the opacity* of a cataract. After an iridectomy for this purpose the lens should be kneaded by massaging the cornea with a cataract-spatula.

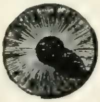
The **instruments** necessary to perform an iridectomy are:

a spring speculum, to keep the lids apart; a fixation-forceps, to steady the eyeball; a bent triangular keratome, to incise the cornea; an iris forceps, to grasp the iris and withdraw it through the corneal wound; curved scissors for excising the part of the iris withdrawn; and a spatula to replace the iris.

The **site** of the iridectomy depends upon the reason for which it is to be made. When for optical purposes, the lower nasal side should be selected, the coloboma should be narrow, and the excised portion of the iris not too large (Fig. 23).

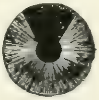
An iridectomy is generally made in the upper part of the iris, since the upper lid will partially cover the coloboma. An iridectomy for glaucoma should be broad and the iris removed near its ciliary attachment (Fig. 24). In order to do this, the corneal incision must be as near as possible to the plane of the iris.

FIG. 23.



Iridectomy downward and inward for artificial pupil. (Nettleship.)

FIG. 24.



Iridectomy for glaucoma. (De Wecker.)

To perform an iridectomy, the operator stands behind the patient, and after the lids are separated by a speculum, the eyeball is steadied with a fixation-forceps at a point opposite the proposed incision, which is then made. The point of the keratome should first be pushed through the cornea perpendicularly, and as soon as it is seen in the anterior chamber the plane of the keratome must be directed from the lens in order that the capsule may not be injured. This should be near the plane of the iris when for relief of tension and in the limbus ordinarily, unless the operation be undertaken for optical reasons, when it may be even inside the limbus. The iris is grasped with the forceps, which have been introduced through the wound, and withdrawn. With the scissors this part is excised, and after replacing the cut edges of the iris, if necessary, the operation is finished. The eye should be kept bandaged for about a week.

Iridotomy is the operation for dividing the iris without removing a part of it. Its application is limited, and then it is not very successful. Most cases requiring it are eyes which are blinded from exudate after cataract-extraction. It may be done with a Graefe knife, which is first pushed through the cornea, when the iris and opaque membrane may be so incised that the pupil becomes a slit.

Sclerotomy is an operation for relieving the increase of intraocular tension. To perform this, a spring-speculum, fixation-forceps, and a Graefe knife are necessary. Before operating the pupil should be contracted by eserine, which is even indicated in glaucoma, in order that the iris may not prolapse. The eyeball being fixed, the Graefe knife, the sharp edge upward, is pushed through the sclera near the corneal margin as close as possible to the plane of the iris on the upper temporal side ; and after crossing the anterior chamber should leave the eye at a corresponding place on the other side. After a few strokes of the knife it should be slowly withdrawn before a flap has been made. A bridge of tissue lies between the incisions, one being on each side. This operation, as a rule, is not so satisfactory as an iridectomy.

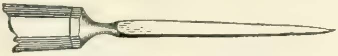
Cataract Operations.

The *linear* and *flap extractions* are the operations for removal of cataracts.

The **linear extraction** is applicable only for soft or membranous cataracts. Before performing this operation the pupil should be dilated with atropine. For a soft cataract a small opening about one-fifth of an inch wide is made with a keratome within the margin of the cornea at its lower border. The capsule is then opened, if this has not already been done by a previous operation, and all pieces of lens-substance are forced out of the eye by pressing on the lower part of it. To remove cataractous membranes after making the corneal opening, they should be drawn out with a hook or forceps.

For removing hard cataracts the **flap extraction** is necessary: The *first step* of the operation is the incision, which should be large enough to give exit to the hardened lens. This is best done with Graefe's straight-knife (Fig. 25), of which the cutting-edge, directed upward, is pushed through the limbus at the upper temporal side, and after passing through the anterior chamber should come out of the eye at a corresponding point on the nasal side. By making a few strokes with the knife the incision is completed, and it is well to include a bit of conjunctiva in the flap.

FIG. 25.



Graefe cataract-knife.

The *second step* is an iridectomy, and the *third* is the opening of the anterior capsule, which is done with a cystitome; and the *last step* is the expulsion of the lens.

After this any extraneous material in the anterior chamber is to be kneaded out by stroking the eye with a spatula, the iris is to be neatly replaced if necessary, the conjunctival wound nicely coapted, and the eyes bandaged.

The extraction may also be performed without an iridectomy, but the beginner will do better by employing the other method.

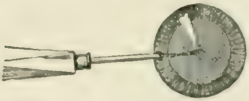
The **instruments** necessary to perform an extraction of a hard cataract are the same as required for an iridectomy (except that a Graefe knife is used instead of a keratome); a cystitome, to tear the capsule; and a scoop.

After extraction both eyes are to be bandaged, and the patient should remain in bed in a dark room for a week. In the meantime the lids are to be gently bathed two or three times daily after the second day with warm borated water, and a drop of atropine instilled at least once daily. At the end of a week the eyes need not be bandaged except at night, and after the third week the patient may be allowed to go out.

Soft cataracts may also be removed by **solution** or by **suction.**

The operation of **discission** causes absorption of the lens-substance by exposing it to the action of the aqueous. To perform this operation, the pupil must be dilated, general or local anæsthesia (according as the patient is unruly or not) employed, and after separation of the lids a cataract-needle is thrust into the cornea near the lower and temporal border (Fig. 26).

FIG. 26.



Discission of cataract. (Nettleship.)

The point of the instrument is pushed through the capsule into the lens-substance, and after this has been gently disturbed the needle is withdrawn. The danger of the operation lies in the possibility of wounding the iris, and this is to be kept in mind. The *after-treatment* consists in rest, use of atropine, and, if there be considerable reaction, application of iced water and leeches.

For removal by **suction**, a cataract must be liquid. A discission is first performed and the nose of the syringe is passed through an opening in the cornea made with a keratome. When the end of the instrument is within the capsule, the contents can be removed by suction. The *after-treatment* is the same as for discission.

THE EAR.

ANATOMY OF THE EAR.

THE ear is divided physiologically into **two parts**, viz.: (*a*) the conduction apparatus, by means of which sound-waves are carried to the terminal filaments of the auditory nerve; and (*b*) the perception apparatus, by means of which sound-waves are received and the impression conveyed to the brain-centres, where they are recognized as sound.

FIG. 27.

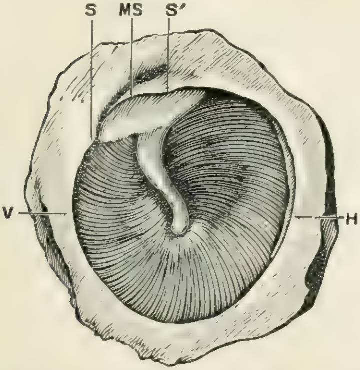


FIG. 28.

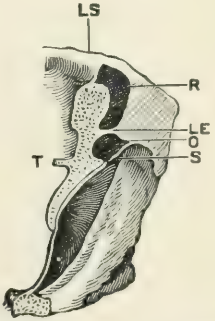
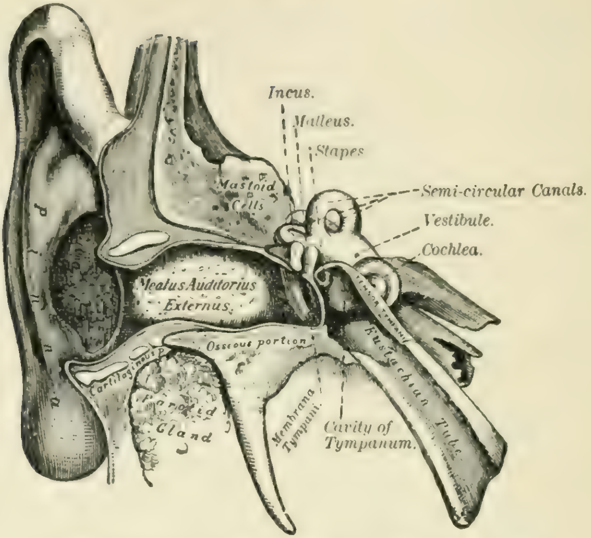


Fig. 27.—Outer surface of the left tympanic membrane of an adult; V, segment of the tympanic membrane lying in front of the handle of the malleus; H, posterior segment of the tympanic membrane: S, S′, Prussak's striæ, passing from the short process of the malleus to the spina tymp. post. et minor; MS, Shrapnell's membrane. (Politzer.)

Fig. 28.—LS, superior ligament of the malleus; LE, external ligament of the malleus, S, Shrapnell's membrane, or the membrana flaccida; O, Prussak's space; R, part of the attic; T, tendon of the tensor tympani muscle. (Politzer.)

The **conduction apparatus** is composed of:

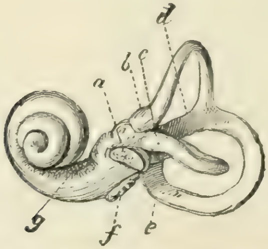
FIG. 29.



A front view of the organ of hearing. Right side. (Gray.)

(*a*) The *external ear:*
1. The auricle, or pinna ; and
2. The external auditory canal (Fig. 29).

FIG. 30.



External view of a cast of the left labyrinth : *f*, fenestra cochleæ, or round window; *a*, fenestra vestibuli, or oval window; *b*, ampulla of superior semicircular canal; *e*, ampulla of posterior semicircular canal; *d*, common shaft of union of these two canals; *c*, ampulla of the horizontal semicircular canal; *g*, tractus spiralis foraminosus. (Henle.)

(*b*) The *middle ear*, or *tympanum:*
  1. Membrana tympani, or drum-head (Fig. 27);
  2. The middle-ear cavity and attic (Fig. 28);
  3. The ossicles (malleus, incus, and stapes);
  4. Eustachian tube (Fig. 29);
  5. Antrum;
  6. Mastoid cells.

The **perception apparatus**, or **internal ear:**
(*a*) The vestibule;
(*b*) The semicircular canals;
(*c*) The cochlea.

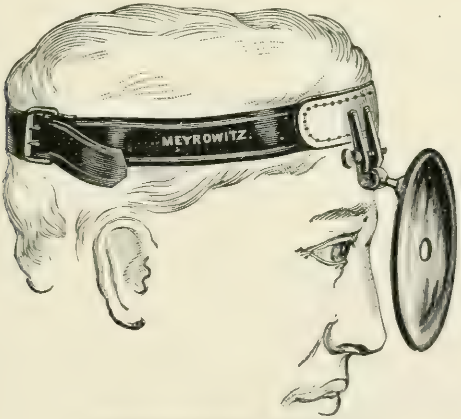
Further anatomical details are given in Figs. 29 and 30.

EXAMINATION OF THE EAR.

Ocular Inspection of the Ear.

The ear should first be examined by the **unaided eye** for malformations and deformities of the *auricle* and *external meatus*. The *mastoid process* should also be examined, and its color and size compared with those of the opposite side. If *scars* exist, they should be noted, and inquiry made as to whether they are due to accidental or surgical injury, or to

FIG. 31.



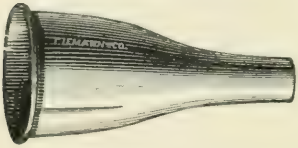
Indirect illumination of the ear.

some disease-process. The auricle and external meatus should be examined for evidences of irritation, such as *excoriations* and *eczematous processes* resulting from middle-ear discharge.

Inspection with the Aid of Reflected Light and the Ear Speculum (Figs. 31–34).

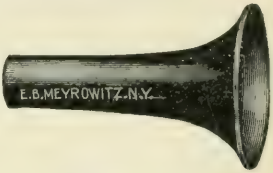
The **external meatus** and **drum-head** should be carefully inspected. The deeper portions of the meatus should be searched for accumulations of cerumen, epithelial scales, and inflammatory areas. If there is a history of mastoid pain, the upper and posterior portion of the external meatus should be especially observed for redness and bulging, as these are signs of active inflammation of the mastoid cells. Eczema of the superior and posterior walls of the meatus is often associated with chronic catarrhal otitis media.

FIG. 32.



Gruber's speculum.

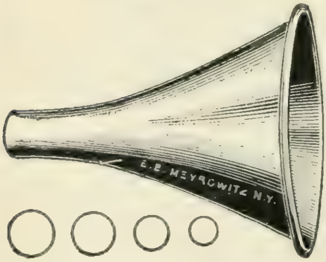
FIG. 33.



Politzer's speculum.

The drum-head should be inspected, and the following conditions noted: The color should be a pinkish, pearly gray. If it is of a whitish, glistening color, with here and there thickened areas, the presence of sclerosis should be suspected. If it is of a dull, sodden, lustreless, pinkish color, otitis catarrhalis is probably present. If it is of a reddish-blue color with a velvety surface, it is indicative of acute otitis media; while if it is of a beefy-red color, with greenish or yellowish areas in the lower half, an acute suppurative otitis media is in active progress.

FIG. 34.



Boucheron's speculum.

It should be noted whether the membrane has the *normal* (Figs. 35 and 36) or an *exaggerated concave surface*, or is *bulging outward.* When the concavity of the drum-head is exaggerated the handle of the malleus is rotated inward and backward, thereby making the short process stand out quite prominently as a whitish, glistening spot in the upper field of the drum-head. The cone of light (Figs. 35 and 36) which projects anteriorly from the umbo, or lower end of the handle of the malleus, is broken or altogether wanting when extreme retraction is present.

FIG. 35.

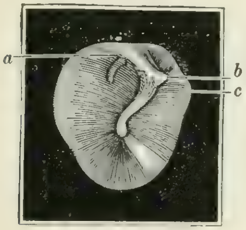


FIG. 36.

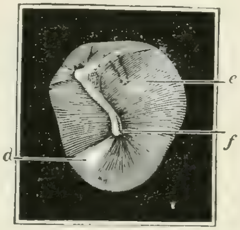


Fig. 35.—Normal drum-head (right ear): *a*, posterior fold; *b*, short process; *c*, anterior fold. (Politzer.)

Fig. 36.—Normal drum-head (left ear): *d*, cone of light; *e*, long handle of incus; *f*, umbo. (Politzer.)

Along the line of the handle of the malleus the *bloodvessels* may be quite prominent, and it should be determined whether this prominence is abnormal or due to the presence of the ear-speculum within the meatus. After the speculum has been in the meatus for a few minutes, the manubrium, which at first had the normal appearance, becomes deeply congested; hence it is important that it should be examined immediately after inserting the speculum.

If the drum-head is *perforated*, the size, location, and character of the edges should be noted; the presence or absence of granulations, polypi, and necrotic bone should be determined.

Examination by Touch.

The **auricle** should be examined by the sense of touch for the presence of calcareous, fibrous, or other hard formations.

The **mastoid region** should also be pressed upon and percussed to see if there is pain or tenderness.

The **external meatus** and **middle ear** should be examined with a probe for evidences of bare bone. If it is present, the probe will impart a grating sensation to the fingers of the operator.

Examination by Smell.

The **probe** introduced into the middle-ear cavity, especially if it is curved and introduced upward into the *attic*, may be the means of demonstrating the presence of a necrotic or **foul odor**, when it would otherwise not be suspected. The probe thus affords another valuable means of diagnosis, in addition to the grating sensation produced by contact with bare bone. It is my invariable custom to note the odor of the probe or dressing removed from the ear. In this way I am enabled not only to arrive at a more correct diagnosis, but also to estimate the progress being made under treatment.

Examination by Means of Instillations.

Sometimes it is impossible to get a view of the drum-head so as to determine the presence or absence of perforations. The **instillation** of *hydrozone* or *pyrozone* will aid in arriving at a conclusion. The drugs cause the pus to break up and give off a gas which imparts a soap-suds appearance to the fluid. This should not be taken, however, as a positive sign of perforation, as an acute dermatitis or furunculosis of the external meatus might give rise to the same phenomenon.

Alcohol or *silver nitrate* may also be used to demonstrate the presence or absence of perforation, by putting a few drops into the external meatus and applying air-pressure through a suitable meatus-tip and forcing the medicament into the middle ear, and out through the Eustachian tube into the throat, where it is detected by the patient.

Examination by Means of Vapors and Air.

Nebulized medicaments may be forced through the middle ear into the throat and the cloudy vapor noted there by the observer, or tasted by the patient. The nebulized vapor may be forced in the opposite direction by means of the Eustachian catheter, or by Politzerization, in which case the nebulous smoke may be seen to issue from the external meatus. A whistling or bubbling sound will also be present if there is perforation. It may also be demonstrated by inflating the ear by Valsalva's or Politzer's method, and noting the hissing sound of the air as it issues through the diagnostic tube (Fig. 42).

Examination by Transillumination.

The condition of the **walls of the external auditory canal** may be studied by placing a hooded electric lamp over the mastoid process. This procedure should be carried on in a dark room, preferably one with blackened walls. If the mastoid cells are free from fluid or other morbid material, the light will be readily transmitted through the bone to the external auditory meatus, rendering it a beautiful, translucent pink color. If there are small areas of bone-necrosis or pus-foci, they will appear as darkened spots against the transilluminated meatus-walls.

The condition of the **mastoid process** may also be studied at the same time.

I have observed, in **chronic suppurative diseases of the middle ear**, that the transillumination is often much diminished upon the side affected. This is due to the pathologic changes in the bone, to hypertrophy of the mucous membrane, and to the presence of pus within the mastoid cells. I have found this means of diagnosis of great value in deciding whether or not a given case of chronic suppurative otitis media should be treated surgically or by the usual classic methods.

FUNCTIONAL TESTS OF HEARING.

The Voice.

This is one of the oldest as well as most practical methods of testing the hearing, as the ear is used more for interpreting the **human voice** than for any other purpose. No matter what amount of improvement may be indicated by the other tests of hearing, the power of the patient to understand the conversational voice must be the final and crucial test. All other tests are of value more in establishing the differential diagnosis than for estimating the amount of improvement in hearing.

Whispered conversation and **numerals** may be used in cases of moderate deafness to establish the distance at which they may be heard. The normal ear will distinguish *whispered conversation* in a quiet room at a distance of from 10 to 30 feet. *Whispered numerals* will be heard at a distance of from 8 to 40 feet, some words and some numerals being heard at a greater distance than others, according to the predominance of vowel and consonant sounds in the spoken words.

Method: The patient should be seated at one end of the room with the ear to be tested turned toward the physician. He should be requested to close his eyes and repeat the words or sentences spoken to him; or if the watch or acoumeter is used, to indicate by raising the hand when he distinctly hears it. The surgeon should approach from the normal hearing distance, using the voice as he approaches step by step. When he has determined at what distance the patient can hear the whispered voice, he should record it in feet or inches.

The **conversational** and **loud voice** may be used in the same way. In extremely deaf persons it is sometimes of advantage to test the hearing through a *speaking-tube* or other aid to hearing. A careful record should be kept of all these tests, so that they may be used for comparison at subsequent times.

The Watch.

The same general rules apply to this test.

Two watches should be used, one a high-pitched and the other a low-pitched ticker. They should be tested upon twenty or more normal ears, so as to establish the normal distance at which they should be heard. The ordinary watch is heard at a distance of from forty to sixty inches.

Having determined the normal distance, it should be taken as the denominator, the numerator being the actual distance at which it is heard. If, for instance, the denominator of the watch is fifty inches and the distance at which it is heard by the patient is ten inches, the record should be Watch = $\frac{10}{50}$.

If the watch is heard only upon *contact against the ear*, it should be recorded Watch = $\frac{c}{50}$; while if it is not heard by either air or contact it should be recorded Watch = $\frac{0}{50}$.

The object of having a high- and a low-sounding watch is of value in differentiating middle-ear and labyrinthine disease; the high ticker being heard best in middle-ear disease, and the low ticker in labyrinthine.

The Acoumeter.

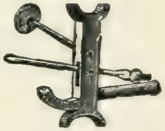
This is an instrument devised by Politzer for the purpose of giving to the specialist a **ticker** tuned to a certain pitch and intensity, whereby it is possible for the records of all observers to be uniform and easily compared.

This instrument can **normally** be heard in a quiet room at a distance of about forty feet (Fig. 37). This should be taken as the denominator and the actual distance at which it is heard as the numerator.

Thus if it is heard at *eight feet*, it should be recorded P. Ac. 8/40.

There is an attachment by means of which the instrument can be placed in contact with the mastoid process and the **bone-conduction** thus tested.

Fig. 37.



Politzer's acoumeter.

The Galton Whistle.

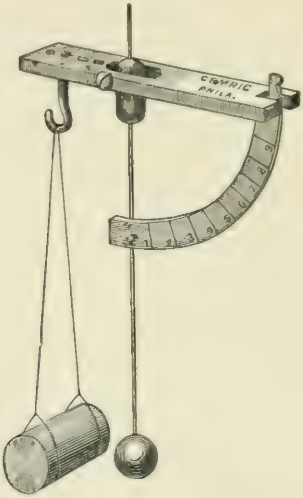
This is an instrument, the **cylinder** of which can be lengthened or shortened by a simple contrivance and the pitch of the tone varied accordingly.

Its **range** is from the highest pitch that can be distinguished by the human ear (about 40,000 vibrations per second) down to near the medium range.

Its **purpose** is to test the *upper limit* of hearing. It is of very great value in the diagnosis of labyrinthine disease, or disease of the auditory nerve where the upper limit of the hearing is considerably lowered.

Konig's rods (Fig. 38) may be used for the same purpose, although they are neither so convenient nor inexpensive as the Galton Whistle.

Fig. 38.



König rod as modified by Blake.

Tuning-forks.

The **vibrations** of the tuning-fork should range from 16 to 4096, selected as follows:

c −3	c −2	c −1	c	c + 1	c + 2	c + 3	c + 4	c + 5
16 v.,	32 v.,	64 v.,	128 v.,	256 v.,	512 v.,	1024 v.,	2048 v.,	4096 v.

With these forks the lower and middle range of hearing may be tested, as well as the comparative hearing-power by air and bone conduction. For ordinary diagnostic purposes the Hartman set of five forks are all that are required. They are as follows:

c	c + 1	c + 2c	+ 3	c + 4
128 v.,	256 v.,	512 v.,	1024 v.,	2048 v.

The upper limit of hearing should be tested with the Galton whistle.

The **usual tests** made by them are the *range of hearing;* and the Weber, and Rinné experiments.

The Range of Hearing.

This is determined for the purpose of ascertaining the lowest and the highest musical tone that can be heard by the affected ear, the opposite ear in the meantime being firmly closed.

The **test** should be made by beginning with the highest fork, and so on down to the lowest that can be heard. If, for example, all the forks from 4096 v. down to 128 v. can be heard, while the 16 v., 32 v., and 64 v. fork cannot be heard, the record should be 4096 to 128 v.

The **significance** of this test is in the fact that in disease of the conduction apparatus the power to hear low tones is diminished.

In cases of extreme middle-ear deafness the patient may not hear lower tones than 2048 v., while he may distinctly hear the notes of the Galton whistle. In labyrinthine disease the upper notes of the Galton whistle are lost, while the low-sounding tuning-forks may be heard.

The Weber Experiment.

This experiment is made for the purpose of determining the **relative hearing of the two ears by bone conduction.**

It is based upon the **principle** that in disease or obstruction of the conduction apparatus bone conduction is increased; while in disease of the auditory nerve or labyrinth bone conduction is diminished.

The **experiment is made** by placing the tuning-fork (preferably the 256 v.) in firm contact with the median line of the head. The fork may be placed on the vertex, forehead, teeth, or chin. The patient is asked to notice with which ear he hears the tuning-fork loudest. If he has disease of the middle ear or external auditory meatus, bone conduction will be increased on that side, while the opposite side remains unaffected.

If, then, the patient complains of deafness in the right ear, and the Weber test shows that he hears the fork best on that side, it is fair to presume that he has disease of the conduction apparatus rather than labyrinthine disease. If, however, he hears the fork best in the normal ear, it is a fair presumption that he is affected with labyrinthine or nerve deafness. Judgment should be reserved, however, until all tests and diagnostic signs are noted.

The Rinne Experiment.

This test is for the purpose of determining the **relative hearing-power by bone and air conduction.**

Normally the forks should be heard from fifteen to twenty seconds longer by air conduction than by bone conduction; for example, if fork 256 v. is placed against the mastoid process and held there until the patient ceases to hear it, and is then held close to the ear, he should continue to hear it for fifteen or twenty seconds more.

If he has **middle-ear disease**, bone conduction will be increased; hence he will hear it longer than normally over the mastoid process, which will leave a shorter time for it to be heard by air conduction.

If, however, there is **labyrinthine disease**, bone conduction will be diminished or totally lost, while air conduction will remain near the normal; hence the fork will be heard by air more than fifteen or twenty seconds after it ceases to be heard (if heard at all) over the mastoid process.

In middle-ear deafness of **marked degree** the hearing by bone conduction may be increased to such an extent that the fork will be heard longer over the mastoid than by air conduction. In this case the test should begin by holding the fork near the affected ear until it ceases to be heard, and then, placing it against the mastoid process, note being taken of the number of seconds it continues to be heard in this position.

Principles Underlying the Tests of Hearing.

1. The normal range of hearing is from 16 to 40,000 vibrations per second.

2. When the conduction apparatus is diseased or obstructed, the power to hear low tones is impaired or lost.
3. When the perception apparatus is diseased, the power to hear high tones is lost.
4. The normal ear hears about twice as long by air conduction as by bone conduction. That is, a fork heard by bone conduction for twenty seconds will be heard about forty seconds when held close to the ear.
5. When the conduction apparatus is diseased or obstructed, bone conduction is increased and the time left in which the fork should be heard by air conduction is diminished ; or bone conduction may be so much increased that the fork is heard longer than by air conduction.
6. When the perception apparatus is diseased, bone conduction is diminished or shortened, so that the relative time of hearing by air conduction is exaggerated.

Practical Application of Tuning-fork and Whistle tests.

Referring to the *third principle*, we note that when the perception apparatus is diseased the power to hear high tones is diminished or lost ; and we note, in *principle five*, that hearing by bone conduction is increased. When we find deafness in a given ear with loss of hearing-power for high tones and diminished bone conduction in the same ear, it is safe to presume the deafness to be due to labyrinthine, nerve, or auditory centre disease. If, however, a patient presents himself with deafness in a given ear, and the Weber test shows bone conduction increased on that side, and the Rinné test shows that hearing by bone conduction is as long or longer than by air conduction, and that the lower tuning-forks are not heard, while the upper forks and Galton whistle are heard distinctly, it is fair to presume that the case is one of middle-ear or external meatus disease. If, on the other hand, the high tones of the Galton whistle are lost and bone conduction is diminished, the case is probably one of labyrinthine disease.

The normal range of hearing for musical tones being from 16 to 40 ,000 vibrations per second, it therefore follows that any deviation from this rule is a sign of some abnormality in either the conduction apparatus (middle ear and external auditory meatus) or the perception apparatus (labyrinth, auditory nerve, or auditory centre in the brain).

Referring to the *second principle*, we learn that when the conduction apparatus is affected by disease or mechanical obstruction the power to hear low tones is lost or impaired. In other words, if there is moderate or marked middle-ear or external meatus disease or obstruction, the tuning-forks giving 16 to 128 vibrations are not heard, while the tones above this limit are heard distinctly. In cases of more pronounced disease the lower limit of tones heard will ascend still higher in the scale. This test is made by holding the vibrating fork near the ear, beginning with the lowest one and gradually ascending the scale until the patient hears the musical note.

Functional Tests—General Remarks.

Much has been written and spoken against the value of the foregoing tests as a means to accurate diagnosis of ear disease. It is true that no one test affords sufficient data upon which to make a diagnosis. It is also true that all the functional tests of hearing may in exceptional cases not afford sufficient data for a positive diagnosis.

If the above principles and experiments are intelligently comprehended and performed, and are considered in conjunction with the other phenomena presented in the clinical history of the case, a correct diagnosis may almost invariably be made.

It should be noted that the above experiments are made for two purposes; namely: (*a*) to estimate the location of the lesion and amount of impairment of hearing; and (*b*) to determine the relative hearing-power by bone and air conduction.

The latter is for the purpose of differentiating between disease of the conduction apparatus (middle ear and external meatus) and the perception apparatus (labyrinth, auditory centre).

These principles and experiments are presented with the hope that they will enable the student and practitioner to render his work not only more accurate. but more attractive as well.

Influence of Age upon Bone Conduction.

For reasons not well understood, bone conduction is considerably diminished in persons **more than fifty years of age.** This should be taken into account in estimating the value of the Weber and the Rinné tests.

Exceptions to the Weber and Rinne Tests.

Many cases in actual practice have at the same time disease of the external, middle, and internal ears, thereby modifying perception by both bone and air conduction.

Bone conduction may be increased by the presence of **a plug of cerumen** more than it is decreased by increased tension of the labyrinthine fluid from pressure on the foot-plate of the stapes, or congestion of the labyrinth, so that the Rinné test may show a predominance of bone conduction over air conduction. Thus the disease of the internal ear is masked. After the removal of the plug of cerumen bone conduction is diminished below the normal limit, thus unmasking or revealing the internal ear disease.

In **suppurative disease of the middle ear** there is often an increase in bone conduction to such an extent as to exceed air conduction; hence when internal-ear disease is combined with it the influence of the internal-ear disease may predominate and show diminished bone conduction, which fact, if taken alone, would not call attention to the coexisting disease of the tympanum.

Boiler-makers suffer from impairment of the nerve-endings in the labyrinth; hence have diminished bone conduction. They may at the same time suffer from pronounced middle-ear disease, whereby bone conduction is increased, thus again bringing the conflicting signs in opposition.

When the physiologic tests, history, course, and symptomatology are given due consideration, the apparent contradictions will become clear and intelligible, and a correct diagnosis be made.

MIDDLE-EAR INFLATION.

Purposes of tympanic inflation: Inflation of the middle ear is practised (*a*) to determine the permeability of the Eustachian tube; (*b*) to aid in arriving at a correct prognosis under treatment; (*c*) to determine the presence or absence of a perforation of the drum-head; (*d*) to clear the Eustachian tube and the middle ear of pus and epithelial *débris;* (*e*) to restore a retracted drum-head to its normal position; (*f*) to restore normal intratympanic pressure, and thereby relieve pain and engorgement; (*g*) to relieve undue pressure upon the foot-plate of the stapes, and thus relieve undue intralabyrinthine pressure; (*h*) to overcome ankylosis of the ossicles; and (*i*) to break down adhesive bands.

The **three methods** in most common use are (*a*) catheterization; (*b*) Politzerization; and (*c*) Valsalva's method of inflation.

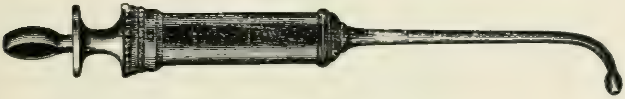
Catheterization of the Middle Ear.

Instruments: This method is accomplished by the use of a silver or vulcanized rubber tube, suitably curved at one end, and having a funnel-shaped opening at the other for attachment to the Politzer bag, or to the shut-off of the compressed-air receiver.

The tubes made of soft or virgin silver are preferable, as they can be bent to suit individual cases. Those made of hard rubber can only be bent under hot water. They possess, however, two advantages over silver; namely, they are not affected by the fluids injected through them, and, as they are flexible they can be more easily insinuated around obstructions in the nares.

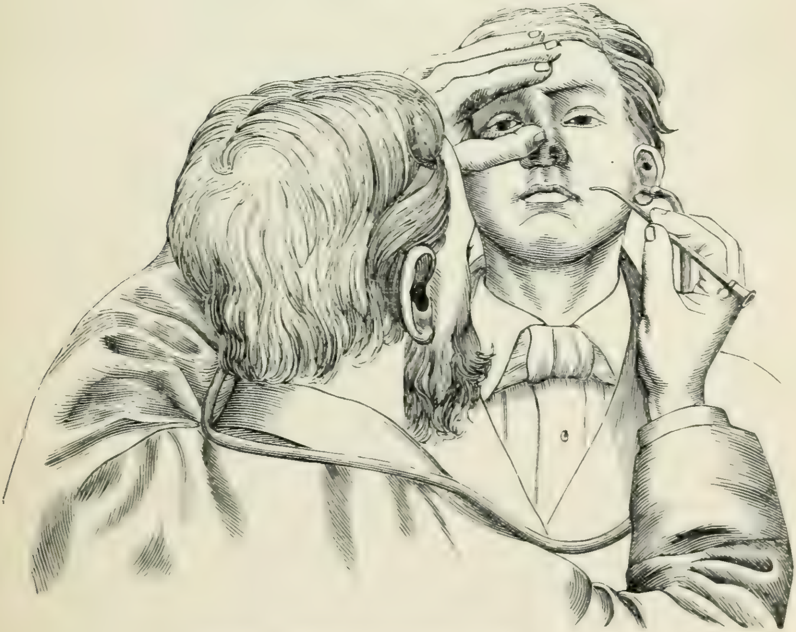
Preliminary to catheterization: (*a*) Carefully examine the nares and postnares for obstructions and diseased conditions. (*b*) Asepticize the nares (anterior and posterior) by the use of postnasal and anterior nasal douches or sprays. The postnasal syringe (Fig. 39) is a convenient instrument for cleansing the postnares. (*c*) Cocainize the nasal and nasopharyngeal mucosa with cotton mops introduced through the anterior nares. This precaution will not only render the procedure painless, but will also contract the mucosa at the mouth of the Eustachian tubes, thus facilitating the entrance of air into the tympanum. One with delicate manipulative technique and large experience will rarely need to observe this precaution. (*d*) Pass a current of air through the catheter to free it from water or other obstructions. (*e*) Place one end of the *diagnostic tube* in the external meatus of the ear

FIG. 39.



Postnasal syringe.

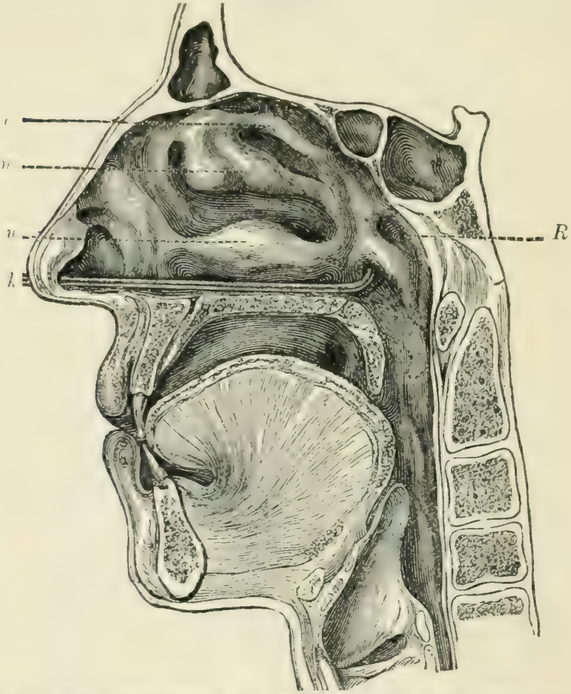
FIG. 40.



Insertion of the Eustachian catheter. (Burnett.)

to be inflated and the other in your own ear. (*f*) Steady the patient's head with the left hand, the index- and second fingers grasping the bridge of the nose, while the thumb elevates its tip; sterilize the catheter.

FIG. 41.



Inner view of the right half of the head: antero-posterior section (Gruber): *o*, superior turbinated bone: *m*, middle turbinated bone; *u*, inferior turbinated bone: *R*, Rosenmuller's fossa, bounded in front by the cartilaginous lip of the tube, in front of the latter is the pharyngeal opening of the Eustachian tube, in which the catheter *k* is placed. (Burnett.)

Introduction of the catheter: *Politzer's method:* The catheter is introduced with the right hand, the curved tip being turned downward and inward on the floor of the nose at its junction with the septum (Fig. 40). It should be gently but quickly passed backward until it touches the posterior wall of the nasopharynx. It should then be turned outward and upward into Rosenmüller's fossa, in front of which is a rounded prominence forming the posterior wall of the mouth of the Eustachian tube. In other words, there is a prominence lying between the end of the catheter and the mouth of the Eustachian tube. The catheter should be held firmly, but with "**a-readiness-to-yield**" to the contour of the surface over which it is to be drawn; with this intention in mind, it is drawn forward over the prominence and then turned outward and upward into the depression or mouth of the Eustachian tube (Fig. 41).

The eyelet upon the outer end of the catheter corresponds to the direction of the curved tip, and should point toward the auricle or outer canthus of the eye.

Gruber's method consists in introducing the catheter as described in the preceding paragraph, and then withdrawing it, curved end downward, until the tip presses against the soft palate, when it should be rotated outward into the mouth of the tube.

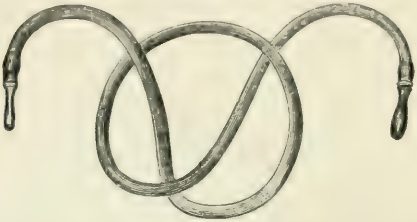
Lowenberg's method consists in turning the curved end of the catheter inward toward the opposite side and withdrawing it until it engages the posterior end of the septum. It is then rotated downward and outward, describing a half circle, into the mouth of the Eustachian tube.

This and the *Politzer* method are the most reliable, as they have fixed anatomical guides—*i. e.*, the posterior end of the septum and Rosenmüller's fossa; while Gruber's plan depends upon the soft tissue. When one method fails, the other should be tried and will usually be attended with success.

After introduction of the catheter: Attach the *Politzer bag* or the *shut-off* of the compressed-air chamber to the outer end of the catheter and force air through it into the Eustachain tube and middle ear. By means of the *diagnostic tube* (Fig. 42) the operator may know whether or not air is entering the middle ear. There will be a soft blowing or bubbling sound, if it is properly gaining admission. The membrana tympani may sometimes be heard to snap as it returns to the normal position. After several interrupted blasts, the catheter should be removed, first turning the curved tip downward.

Dangers attending the use of the catheter: The mucous membrane may be friable, or unskilful manipulation of the catheter may cause it to penetrate the epithelial covering of the mucosa. In either event inflation will be followed by emphysema of the submucous tissue of the neighboring parts, as the uvula, cheek, soft palate, or throat. A few cases have been reported in which there was a fatal issue from suffocation. The skilled physician need have little fear of producing such a result. The use of high pressure from a compressed-air tank is to be condemned on account of this danger. If the air-tank is used, the gauge should not register more than seven to fifteen pounds. For most purposes it is better to use the Politzer bag, as with it there is little probability of causing emphysema.

FIG. 42.



Auscultation-tube. Black end for patient's, white for surgeon's, ear,

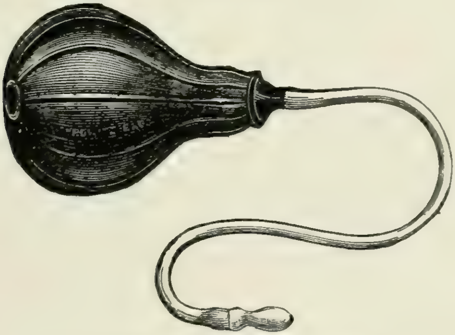
Politzerization of the Middle Ear.

Instruments: This very useful procedure is done with a *soft-rubber bag* (Fig. 43), of about eight ounces capacity. To the hard-rubber fitting at the smaller end of the bag is attached a piece of *rubber tubing*, at the other end of which is a hard-rubber *nasal-tip* for application to the anterior nares.

General method: The nasal tip should be inserted into one of the anterior nasal openings, the opposite nostril closed, and air forced into the nasal chambers by a sudden and forcible compression of the bag with the disengaged hand.

Unless **certain precautions** are observed, the air will escape into the pharynx and œsophagus instead of going into the middle ear. There are *three methods* in common use which are more or less successful in rendering inflation by the Politzer method effective, namely: (*a*) Instruct the patient forcibly to inflate the cheeks, thereby causing the palatine arch to separate the nasopharynx from the lower pharynx. (*b*) Another method consists of pronouncing certain sounds, which momentarily shuts off the communication between the nasopharynx and pharynx, and at the same time opens the mouths of the Eustachian tubes. The sounds best adapted for this purpose are the english "k," or the german "och." At the moment of pronunciation the bag should be forcibly compressed. (*c*) The third and the most successful method requires the patient to take a sip of water in the mouth and retain it there until told to "swallow," and at the height of the act of swallowing the physician compresses the bag as in the other methods.

FIG. 43.



Politzer bag, with glass nose-piece.

By any one of these methods air is shut off from the lower air-passages, the mouths of the Eustachian tubes are opened, and air is allowed to pass into the middle-ear cavity. If, however, there is considerable thickening of the mucosa of the tubes, or if there is a *stricture* or fibrous band closing the tubes, it may be necessary to resort to the use of the catheter, and in extreme cases to the Eustachian bougies.

Valsalva's Method of Inflation.

This method is mentioned chiefly for the purpose of condemning it. It is performed entirely by the patient, and therein lies the danger.

Method: The patient should close both nostrils with the thumb and index-finger, take a deep breath, and forcibly attempt to expel the air through the closed nostrils. The method is pernicious because of the ease with which the patient can resort to it, and because inflation when continued over a protracted period results in atrophy of the drum-head.

I have seen cases in clinics in which indiscriminate inflation had been done for six or more months, and in which the drum-head was thin and parchment-like in appearance.

As a rule, it is well to limit the inflations to a period of about six weeks. If good results do not follow in this time, it is useless to continue such treatment longer.

STRICTURE OF THE EUSTACHIAN TUBES.

Etiology: Long-continued catarrhal inflammation of the mucosa of the tubes may be attended by the formation of fibrous bands in the submucous tissue. After a time they become contracted and encroach upon the lumen of the tubes.

Symptoms: The normal supply of air to the middle-ear cavity is thus interfered with, and the drum-head becomes retracted in consequence. This condition, of course, is attended by tinnitus and deafness.

Treatment.

The **stricture should be dilated** by the introduction of small bougies made of whalebone, celluloid, or other flexible material. Bougies of graduated sizes may be introduced in succession or at successive treatments, as in stricture of the urethra. The treatments should not be repeated oftener than three times a week, but should be continued until the stricture shows no signs of contraction.

Method of introducing the bougies: (*a*) First insert a catheter with a lumen large enough to allow the bougie to pass freely. (*b*) Inflate the tympanum, and with the diagnostic tube determine whether or not the catheter is within the Eustachian orifice. (*c*) Introduce the bougie into the catheter until it reaches the Eustachian tube. It should then be carefully and gently pushed into the tube and beyond the stricture, and left there for several minutes.

Extreme care must be observed to avoid forcing the catheter through the mucous membrane. It is well to tie a thread around the bougie at a point marking the length of the catheter, so as to know when it is at the Eustachian orifice. The bougie may be introduced an inch or an inch and a quarter without danger of injuring the contents of the tympanum.

GENERAL ETIOLOGY OF DISEASES OF THE EAR.

(*a*) Most ear diseases have their origin in **affections of the nasopharynx.** (*b*) Perfect hearing depends upon free ventilation of the middle ear.

The two propositions set forth in the above sentences embrace most of the factors which are primarily the cause of ear diseases and disturbances of hearing.

Nasopharyngeal Diseases Affecting the Patency of the Eustachian Tubes.

1. **Acute postnasal catarrh**, as it is usually found associated with acute coryza, closes the Eustachian tubes by congestion of the mucosa at their orifices.

2. **Chronic postnasal catarrh** does the same thing by chronic swelling and hypertrophy of the mucosa.

3. **Hypertrophy of the faucial tonsils** interferes with the action of the tensor palati muscles (which lie just anteriorly and inferiorly to the Eustachian orifices), and with the levator palati, which take origin partly from the cartilages of the tubes. As the muscles control the patency of the tubes, it is quite evident that enlargement and adhesions of the tonsils to the pillars of the fauces play an important rôle in the etiology of ear disease.

4. **Postnasal adenoids** may interfere with the action of the tensor and levator palati muscles; but they are chiefly of

interest in this connection because they sometimes overlap the ostium tubæ, thereby interfering with ventilation. Pus may gain entrance into the tube and middle ear and excite a suppurative inflammatory process.

Nasal Diseases Affecting the Hearing.

1. **Acute and chronic rhinitis** are attended by engorgement of the mucosa of the nasopharynx. This process may extend into the tube and ear, and become established there as chronic otitis media catarrhalis and tubal catarrh.

2. **Nasal polypi** obstructing the respiratory tract of the nose cause engorgement of the mucosa about the tubes.

3. **Hypertrophy and swelling** of the posterior ends of the **inferior turbinated bodies** also excite thickening at the Eustachian orifices.

4. **Deflections, enchondroses, exostoses,** and other **thickenings of the septum** are also important factors in producing similar results.

Miscellaneous Causes of Ear Disease.

1. **Microorganisms** peculiar to the pharyngeal inflammations attending specific fevers may gain entrance through the Eustachian tube into the middle ear and excite very active suppurative disease of the same. Suppurative inflammations following scarlatina, diphtheria, and influenza are particularly destructive. The suppurations thus excited often become *chronic*, destroying in many instances the entire drum-head.

2. **Paralysis of the palatine muscles,** following the specific fevers, interferes with the patency of the Eustachian tubes.

3. **Vomiting and catheterization** may be the means of carrying infection into the middle ear.

4. The primary source of inflammation may be transmitted **through the lymphatics** from an erysipelatous inflammation of the face and scalp.

5. **Inflammation** of the **auditory canal,** or of the **cranial cavity,** or of the **labyrinth** rarely extends into the middle ear. *Labyrinthitis* may be caused by infections from the cranial cavity by way of the æqueductus vestibuli into the vestibule.

Sequelæ of middle-ear inflammation: The process may extend into the *antrum* and *mastoid cells*, and by *necrosis* into the *cranial cavity*. It also sometimes extends into the internal ear, thereby causing *nerve deafness*.

Meningitis and *pyogenic diseases* of the *brain* almost always arise from pyogenic disease of the middle ear and mastoid.

Paralysis of the facial nerve sometimes arises in the course of chronic suppurations of the tympanum. It may be due to necrosis of the bony covering of the nerve, or to a congenital absence of such covering.

Chronic catarrhal inflammation of the middle ear renders the *ossicula* and foot-plate in the oval window more or less *rigid*. The incudostapedial articulation, round window, and stapedovestibular joint are thereby somewhat ankylosed. In the later stages of the disease *true sclerosis* of the parts develops, with consequent increased rigidity.

Anatomo-physiologic Considerations.

The function of the **tensor tympani muscle** is to control the tension of the drum-head. It is the focusing muscle (with the stapedius) of the ear, enabling it to select at will sound-waves or voices pouring in upon it at the same time. The nerve-supply is from the motor branch of the fifth (possibly from the facial). Electric stimulus applied to the fifth produces strong contraction of the muscle, during which the drum-head is drawn in by the handle of the malleus. During this contraction there is increased tension of the intralabyrinthine fluid.

The **palatal muscles** are stimulated to contraction at the same time, thereby showing a close relationship between the tube and the middle ear.

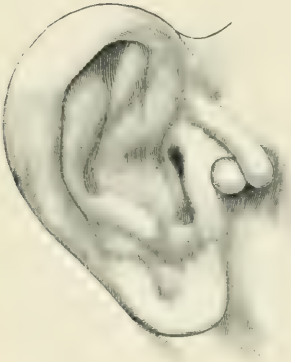
The **stapedius** also contracts under stimulus to the facialis, which suggests that it may act as a check upon the tensor tympani.

Relation of the tensor tympani and stapedius to hearing: In view of the above facts, it becomes apparent that any morbid process affecting the length, mobility, or innervation of these muscles will produce disturbances of hearing. Hence chronic catarrh of the middle ear attended by hyperæmia, hypertrophy, sclerosis, adhesive bands, etc., is characterized by disturbances of hearing, chief among which are deafness and tinnitus.

THE AURICLE.

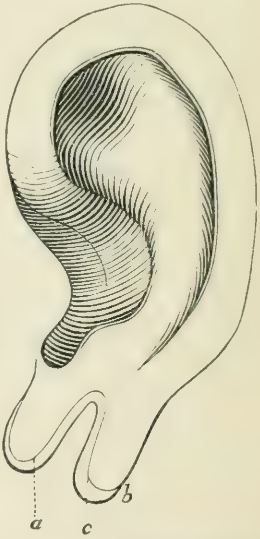
Congenital defects of the auricle: There may be an entire absence, a partial development (microtia), or a misplacement of the auricle. In rare instances it has been located on the cheek. Occasionally there is a blind canal, known as the *fistula auris congenita*, located in the ascending part of the helix and opening either just anterior to the tragus or upon its inner surface at the entrance of the external meatus. It is lined with epithelium and secretes a creamy fluid. It can be obliterated by laying it open with a knife and removing the secreting membrane. Very rarely there is a supernumerary (polyotia) auricle (Fig. 44).

FIG. 44.



Rudimentary auricle in front of the tragus.

FIG. 45.

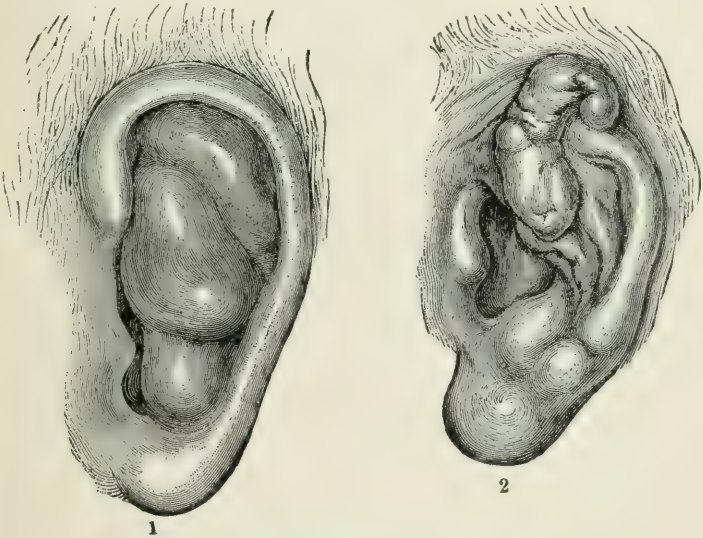


Cleavage of the lobule with edges freshened for surgical reunion.

Injuries to the auricle: These may consist of cuts, bruises, burns, cleavage of the lobule (Fig. 45) from forcible removal of an ear-ring, and sloughing from the presence of a brass ear-ring.

Hæmatoma of the auricle (othæmatoma Fig. 46) may arise from a blow in foot-ball playing and in boxing. It may also be spontaneous, as is often found among the insane.

Fig. 46.



Othæmatoma (1), and the resultant deformity (2). (Gruber.)

In *slight cases* cooling lotions should be applied. In the *severer cases* the tumor should be incised and thoroughly freed from clots and other detritus, after which it should be thoroughly irrigated and sutured, except at its most dependent portion, into which a small wick should be inserted for drainage.

Frostbite: When the auricle is frostbitten, it becomes yellowish-white in color, and in extreme cases may be actually congealed and brittle. The *treatment* should consist in the immediate application of cold and friction for the restoration of the circulation. After this has been accomplished the local application of heat and of the lead-and-opium wash should be made. If there are bluish or inflamed areas beginning to ulcerate, applications of the tincture of iodine or collodion act favorably. If gangrene or sloughing sets in, it should be treated with boric acid or iodoform ointments.

Perichondritis of the auricle is characterized by rapid development, the skin being hot, red or purplish in color, and painful. Upon incising the swollen parts there is a discharge of yellow fluid or pus, and the probe shows the perichondrium to be detached. The exposed cartilage presents a roughened surface when the probe is passed over it. The skin is infiltrated, the swelling often extending into the external meatus, thereby partially or completely occluding its calibre. The disease runs a more acute course than hæmatoma, and the history of traumatism is absent.

When *treated early*, the application of cold and the lead-and-opium wash often checks the progress of the process. In the *later stages*, when fluctuation appears, it should be incised, irrigated, and closed with sutures and a bandage applied. Healing usually occurs by first intention if there is a thorough removal of all morbid material and antiseptic precautions are observed. Some have recommended the withdrawal of the fluid with a hypodermic needle, after which the tincture of iodine should be injected into the cavity.

Cysts of non-inflammatory origin, containing clear fluid, sometimes form on the auricle.

Eczema of the auricle usually occurs in connection with eczema of the external auditory meatus, and will be further considered in that connection.

Lupus of the auricle occurs in connection with lupus of the skin of neighboring parts.

Syphilitic inflammation of the auricle is one of many associated expressions of the syphilitic poison, and should be treated according to the usual indications.

Hyperæmia of the auricle may follow frostbite, hot poultices, eczema, and inflammatory ear diseases. It also occurs in neurotic patients and in valvular disease of the heart.

Diffused inflammations of the auricle may occur as the result of injury, frostbite, heat, stings, and infections.

THE EXTERNAL MEATUS.

Congenital malformations, as dilatation, atresia, entire absence, and double canal are occasionally observed.

Injuries to the external auditory meatus may result from the accidental or intentional introduction of instruments and other hard bodies. Persons affected with "itching ears" often attempt to relieve the disagreeable sensation by scratching the external meatus with a pin or other hard instrument, and sometimes injure the skin in the attempt. Live insects occasionally gain entrance into the meatus, and unskilful attempts to remove them often result in injury. Injuries may also arise from the introduction of hot water, caustic fluids, and the unskilful use of the electric cautery in paracentesis of the drumhead. Hot cinders also occasionally lodge in the meatus and excite inflammation.

Ceruminal Accumulation or Retention.

The plugs are usually **composed** of an admixture of *cerumen, sebaceous material, epidermis,* and *short hairs.*

Etiology: Recurring hyperæmia of the external meatus often leads to the retention of cerumen. Contraction of the meatus and tenacious secretion also favor its retention. The ceruminal glands are located in the cartilaginous portion of the meatus, and the secretion is usually spontaneously discharged. Attempts to remove it with the twisted end of a towel forces the cerumen into the osseous portion of the meatus, where it becomes inspissated and admixed with epithelium and short hairs, and finally completely fills that portion of the canal. Eczema and other inflammations of the external auditory meatus favor ceruminal accumulations. Foreign bodies lodging in the canal act as foci around which the secretion and epithelium gather.

Ceruminal accumulation—symptoms: There may be a feeling of fulness and confusion, with tinnitus and subjective noises. The patient's voice seems very resonant to himself, and he sometimes complains of giddiness and vertigo. In very rare cases there are vomiting and epileptiform convulsions. There is usually dulness of hearing, although when associated with perforation of the drum-head the plug may improve the hearing by acting as an artificial membrane. Conversation is usually understood by the patient. When speech is not understood, there is probably an associated middle- or internal-ear disease. The ceruminal plugs appear as light-yellow or dark-brown, dull, glistening masses. They may be soft to the point of liquefaction or as hard as stone.

Prognosis: Where the plugs are primary or unassociated with disease of the middle and internal ears, the prognosis as to hearing is good. If speech is not understood, there is middle- or internal-ear disease; hence the prognosis for hearing should be guarded. If the Weber test shows bone conduction to be greater in the better hearing ear, there is probably internal-ear disease, and the prognosis as to hearing is bad.

Ceruminal accumulations—treatment: The instrumental removal of the cerumen is attended by considerable pain, and there is danger of injuring the skin of the meatus or forcing the plug against the drum-head, thereby occasioning great disterss and possible injury to the structures of the middle ear. The syringe affords the best and safest means for its removal. A syringe holding from three to four ounces is best adapted to the purpose. If after several forcible injections of water the cerumen is not removed, the ear should be dried and the attempt discontinued. The patient should be given a phial of 15-volume hydrozone and instructed to instil it into the ear four times daily for two days. At the end of this time he should return to the office and the warm-water injections with the syringe be resumed. The instillations of hydrozone will have softened the cerumen, so that it may be easily removed with the syringe. Glycerin and solutions of soda may be used instead of hydrozone; although in my experience hydrozone has served a much better purpose. After the removal of the ceruminal plug the external meatus should be painted with carbolized lanolin or other antiseptic ointment.

Absence of cerumen in the external meatus: This condition is often observed after a diffused or circumscribed inflammation of the external ear, and in the course of adhesive processes in the middle ear. The trophic nerves seem to be affected by these diseases, thereby interfering with the functional activity of the ceruminal glands. The *treatment* consists of mercurial ointments applied at intervals of forty-eight hours and the internal administration of nux vomica.

Furunculosis of the External Auditory Meatus.

Synonyms: Follicular Inflammation; Circumscribed Inflammation and Boils.

Occurrence: Furunculosis of the external auditory meatus is usually limited to either the cartilaginous or the bony portions of the canal. The boils may be localized or associated with general furunculosis.

Etiology: They may arise from frequent syringing of the ear, or from attempts at the instrumental removal of cerumen or foreign bodies. The prolonged use of astringents, as alum-water, has been known to cause them. Streptococcus and staphylococcus pyogenes sometimes enter the deeper layers of skin through the hair-follicles and cause the inflammation. In some cases the cause is unknown. Furunculosis appears more frequently in the spring and autumn; and in adults rather than in children and old people. Anæmia and disturbances of menstruation and digestion, exposure to cold air, and anæmia also favor its appearance.

Furunculosis—symptoms: If the boils are located in the superficial layers of the skin, the pain is slight; while if in the deep layers, it may be quite severe. There are often tenderness and swelling over the mastoid region, which may cause the condition to be mistaken for acute *mastoiditis*. The hearing is more or less impaired and tinnitus may or may not be present. There is often an itching sensation in the meatus, and the patient has an irresistable desire to use a pin for its relief. There is usually a slight febrile movement, especially at night. If the circumscribed inflammation is deep, it appears as a flat, red elevation; if superficial, it is circumscribed, pointed, red or livid, and attended with but slight pain. The pus may discharge spontaneously after eight or ten days. The boils are usually located on the anterior cartilaginous wall.

Furunculosis—treatment: In the deep-seated type free incision should be made to relieve the pain. It is not well to wait for pus to form, as an early incision is often followed by improvement. If the incision is made after the formation of pus, slight pressure should be applied with a probe to assist in the removal of the tenacious pus from the deeper tissues.

In the *early stages*, while there are considerable irritation and pain, the application of cold with the Leiter coil will modify the progress of the disease. For the relief of pain the instillation of a 5 per cent. solution of cocaine and the application of ointments of the acetate of morphine should be used. Hot fomentations of hops applied at frequent intervals is also a usefel remedy.

During the *stage of inflammatory infiltration*, the application of a 3 to 5 per cent. solution of carbolic acid in glycerin often aborts the disease. After the inflammation begins to recede, a 1 : 3000 solution of mercuric bichloride promotes rapid resolution.

After the boils have discharged their contents spontaneously, or by incision, they may be treated by the remedies mentioned, after the application of which the meatus should be thoroughly dried and packed with a narrow strip of gauze. This should not be removed by the patient, but should be left in place and removed by the physician on the following day. The same process should be repeated until the inflammation has subsided. In cases in which there are recurrent attacks of furunculosis there should be appropriate constitutional treatment.

Diffuse Inflammation of the External Auditory Meatus.

Etiology: This type of inflammation may be due to the instillation of irritating drugs, injuries, foreign bodies, and associated mastoid disease.

Symptoms: Pain is present in varying intensity, and is worse at night; and upon movements of the jaw, or fits of coughing. Tinnitus and giddiness are sometimes present. The hearing is normal, except in those cases in which the drum-head is involved. The osseous portion of the canal is hyperæmic. After two to three days there is a discharge, which becomes purulent as the disease advances. In some cases a very thick mucous or gelatinous plug forms, and completely fills the deep portion of the meatus. The secretion may form in layers composed of mucus, pus, epidermic scales, and pyogenic cocci. If it is allowed to remain for two or three days, it decomposes and becomes offensive. After the removal of the secretion the skin beneath is found to be red, swollen, and œdematous. There may be ulceration and excoriation.

Diffuse inflammation of the meatus—treatment: In severe idiopathic cases the local application of cold and leeches reduces the engorgement and checks the process of infiltration. In cases due to traumatism the application of the Leiter coil (Fig. 57) and local asepsis, with gauze packing, is usually followed by a favorable termination. If the disease has advanced to the stage of secretion, antiseptic powders and a saturated solution of boric acid in alcohol should be used. In very obstinate cases, especially when granulations are present, a strong solution of silver nitrate applied locally reduces the granulations and promotes epidermization. Sometimes great relief is obtained by incision at the point of greatest tenderness.

The so-called "dry treatment" (packing the external meatus with strips of sterilized gauze) is perhaps as efficacious as any other. The secretions are absorbed by the gauze, thus keeping the skin comparatively dry during the intervals between treatments. If constitutional dyscrasiæ are present, they should receive appropriate treatment.

Hemorrhagic Inflammation.

It is **characterized** by hemorrhages in the skin of the osseous meatus, rarely in the cartilaginous, and is attended by inflammatory reaction. It usually occurs in the young without known cause, except in cases due to influenza.

Symptoms: Tinnitus, slight pains, and deafness are usually present. Long, dark-blue swellings may be seen on the inferior wall, sometimes extending into the post-inferior quadrant of the drum-head. They are soft to probe pressure and are easily perforated. They contain a bloody serum and attain full development about the third day. Hemorrhagic vesicles usually persist several days later.

Hemorrhagic inflammation of the meatus—treatment: This is usually followed by complete recovery. The epidermis is cast off in large scales in from eight to ten days, after which the parts assume their normal appearance. The bluish swelling should be opened with a probe or knife, after which the external meatus should be irrigated with boric acid solution, dried, and powdered with boric acid. The meatus should then be packed with a strip of sterilized gauze, which should be removed the following day. Three or four such treatments are usually all that is necessary for the complete cure of the cases.

Croupous and Diphtheric Inflammation of the Meatus.

This condition is **rare**, and is limited to the *osseous portion* of the canal and to the drum-head. It usually follows an exhausted attack of faucial diphtheria.

Treatment: The meatus should be irrigated with lime-water. After removal of the membrane instillations of a 10 per cent. solution of carbolic acid in glycerin should be applied with a small cotton mop, to prevent its return.

Otomycosis; Mycosis of the External Auditory Meatus.

This is a **parasitic inflammation** due to the Aspergillus niger, A. flavus, and A. fumigatus (Fig. 47). Occasionally other fungi are also present.

The growths **appear** as brownish or black plaques upon the skin of the meatus, or upon moist crusts. One or two cases have been reported in which the parasites grew in the *middle ear*.

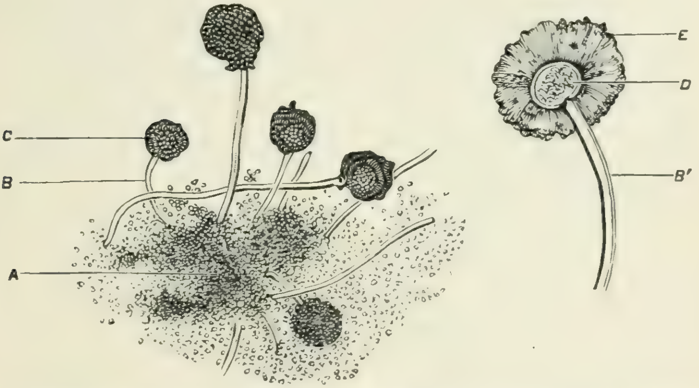
When growing upon moist crusts they may form layer upon layer, until the meatus is more or less filled. They show a tendency to recur at intervals of days or weeks. In doubtful cases the microscope will aid in the diagnosis.

The **treatment** consists in the instillation of alcohol, which stops their growth after one or two applications. The treatment should be repeated once every month for a year, to prevent their return.

Eczema of the External Ear.

Occurrence and varieties: Eczema in this locality differs in no way from eczema on other portions of the skin. It may be *acute* or *chronic*, but is more often of the former type. It may attack all ages and either sex, but is more common in children. It may be *erythematous*, *vesicular*, *pustular*, or *squamous*. It may be limited to the auricle or to the external auditory canal, or it may involve both at the same time.

FIG. 47.



Aspergillus nigricans: A, mycelium covered with numerous fallen spores; B, hypha; C, sporangium with ripe spores; B′, hypha; D, receptaculum; E, sterigmata with spores. (Politzer.)

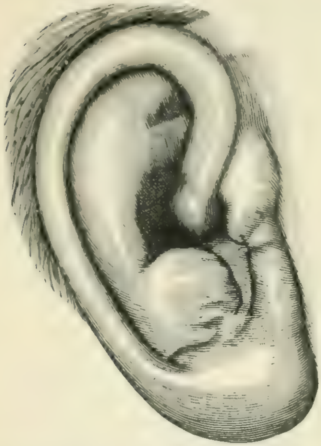
Symptoms: If of the *acute type*, the skin is infiltrated and swollen, and may obstruct the external auditory meatus.

If it is of the *chronic type*, there is permanent thickening of the tissue, which may occasion considerable deformity of the ear (Fig. 48).

A feeling of fulness, moderate deafness, and tinnitus are often complained of.

Eczema of the external ear—treatment: In acute cases the administration of 10 to 20 drops of the fluid extract of Viola tricolor is attended by good results, especially in children. If the patient be of a gouty or lithæmic diathesis, suitable internal treatment should be given. Constitutional dyscrasiæ should be corrected by the administration of the syrup of ferrous iodide, cod-liver oil, and arsenic. In the local treatment, the use of water should be avoided, as it aggravates the condition. If it is of the moist variety, the eczematous areas should be painted with a 10- to 30-grain solution of silver nitrate. If it is of the dry, scaly type, an ointment containing 1 grain of salicylic acid and 4 minims of the tincture of benzoin in 1 drachm of lanolin, applied twice daily, is followed by great relief from the stiffness and excessive epithelial desquamation. After removal of the crusts the meatus should be powdered with equal parts of boric acid and zinc oxide. A 10 per cent. ichthyol or resorcin ointment, locally applied, is also a very useful remedy. Moist surfaces back of the auricle, especially at the union of the auricle and the skin over the mastoid, should be dusted with borated powder or with pulverized calomel.

FIG. 48.



Thickening and deformity of the auricle, due to chronic eczema. (Bacon.)

Seborrhœa.

This condition is usually **associated** with a similar affection of the face and scalp.

Symptoms: When it affects the external auditory meatus, there are itching, tinnitus, and slight deafness.

The **treatment** should consist in removal of the scales, after which the parts should be dusted with equal parts of boric acid and zinc oxide. An ointment of white precipitate is also a useful remedy.

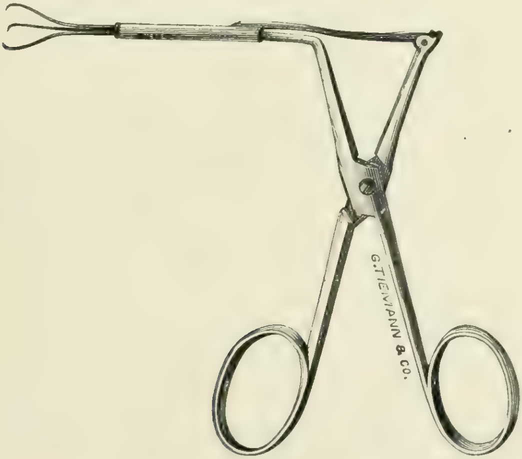
Foreign Bodies in the Meatus.

Varieties: Insects, such as cockroaches, bedbugs, fleas, beetles, etc., sometimes get into the external meatus, and give rise to very distressing symptoms. Wads of cotton, paper, portions of pencils, seeds, beads, cherry-stones, and many other inanimate objects have also been found in the meatus. Beans, wheat, and other grains, which swell upon being exposed to moisture, may give rise to marked *pressure symptoms.*

The **symptoms** in general are those of obstruction to the meatus, attended by more or less inflammation.

Foreign bodies in the meatus—treatment: Do not attempt to remove live bugs from the ear, as the effort may prove fruitless, and occasion great distress to the patient. They should first be drowned in water, oil, glycerin, or alcohol, after which they can be easily removed with the syringe. If this method fails, they may be removed with forceps, or a short-toothed ear-hook. Allen's foreign-body forceps (Fig. 49), and the ear-hook are valuable instruments for the removal of inanimate bodies, as beads, paper, cotton, etc. A

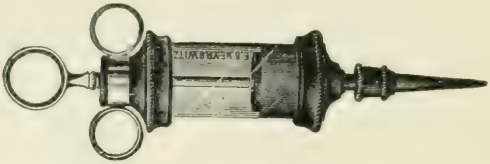
Fig. 49.



Allen's foreign-body forceps.

syringe (Fig. 50) with a flat, thin nozzle, which can be inserted at the side of the foreign body may be used to force water back of it, and thereby drive it outward. If there is perforation of the drum-head, the syringe may be attached to the Eustachian catheter, and water forced through the Eustachian tube and tympanum and outward through the perforation into the meatus, thereby dislodging the foreign body. This method is not practicable if the foreign body is firmly imbedded in the meatus. Smooth, hard bodies, like beads, may be removed by dipping a camel's-hair brush in glue and applying it to the surface of the foreign body, leaving it there until it becomes quite dry. The removal of the brush will bring the foreign body with it.

FIG. 50.



Bacon's ear syringe.

Hemorrhage and Ear-cough.

Hemorrhage from the ear: It may be *due* to direct injury, fracture of the bony canal, vicarious menstruation, and to vascular granulations, as in tuberculosis of the middle ear.

Ear-cough: Foreign bodies and inspissated cerumen may excite an irritation of the auricular branch of the pneumogastric nerve which supplies the external meatus.

Stricture of the Meatus.

Etiology: Stricture of the meatus may be due to swelling of the skin in chronic suppurative otitis media, and other persistent inflammations and irritations. *Bony stricture* (hyperostosis) may follow caries and exfoliation of the sequestra from the osseous walls of the meatus. Septa or adhesions may form across the canal by the approximation of granulations or inflamed surfaces. An *exostosis* is a circumscribed thickening of the bone projecting into the lumen of the canal.

Stricture of the meatus—treatment: When the stricture is due to thickening of the soft tissues of the meatus, it should be irrigated with warm boric acid solution, dried, and a cotton-wool plug, dipped in a 4-grain iodoform ointment, packed firmly in the opening. This should be done twice daily. In the more obstinate and chronic cases small laminaria tents should be introduced every three or four days and allowed to remain for about two hours each time. The patient should remain under the observation of the physician during the treatment, as it is important that too great pressure be avoided. If these methods fail, longitudinal incisions should be made in the meatus and cotton-plugs or sponge-tents applied, as already described. If adhesions or septa are present, they should be divided and distended with cotton-plugs or sponge-tents. Osseous contraction may also be treated in the same manner, provided great care and patience are observed. Associated disease of the middle ear, as chronic suppurative otitis media, should receive appropriate treatment.

Collapse of the meatus: This occurs in the aged, who have developed a flaccid condition of the connective tissue of the skin. Obstructive deafness caused by it may be relieved by wearing a silver tube in the meatus.

Exostosis of the Meatus.

This is a **circumscribed bony growth** projecting from the wall of the osseous meatus, and is usually found in those with a gouty diathesis.

It is more common in males than females. Persons who are much in the water, as the South Sea Islanders, are very liable to be thus affected.

The **treatment** consists in removal with the cold-wire snare or the dental burr. It may also be removed by making a post-auricular incision at the junction of the auricle with the mastoid, and pulling the auricle and soft meatus forward, as for a mastoid operation. The tumor is thus fully exposed, and may be removed with a chisel or a dental burr. The bone beneath it should also be slightly excavated so as to prevent recurrence.

Caries and Necrosis of the Meatus.

Etiology: It is almost always secondary to pyogenic inflammation of the middle ear and mastoid cells.

Diagnosis: The softened carious bone may be detected with the probe. If the bone is necrotic, its hard roughened surface imparts a characteristic grating sensation to the fingers as the end of the probe is passed over it. This condition is usually located in the post-superior osseous wall, which appears, red, swollen and œdematous.

The **treatment** consists in free incision through the swollen area, followed by curettement of the carious bone. In some cases it will be necessary to resort to the radical mastoid operation, as the antrum and mastoid cells may also be extensively involved.

Injury to the Drum-head.

Etiology: Rupture of the drum-head may be caused (*a*) by direct violence, as from some hard penetrating body; (*b*) by injury to the bones of the cranium, as from a blow on the chin; (*c*) by impacted air in the external auditory meatus, from a blow with the palm of the hand upon the auricle; and (*d*) by concussion of the air, as following the firing of a cannon.

Symptoms: The rupture is usually in front of the handle of the malleus, and is covered with a bloody discharge. It usually heals quickly, but may be followed by inflammation, *especially if improperly treated*. The labyrinth may be involved. The bone-conduction and Galton-whistle tests should be used to determine this fact. When it is involved, there are usually giddiness, nausea, and vomiting.

The **treatment** consists in the insertion of a strip of dry sterilized gauze into the external meatus.

Acute Myringitis.

Synonym: Acute Inflammation of the Drum-head.

Symptoms: In the early stage it is characterized by redness of the drum-head in the posterior quadrant, or it may involve the whole surface. The inflammation is often limited to the dermic layer, although it may extend through the fibrous to the mucous layer. It is in such cases that pus-pockets form between the fibrous and mucous layers. The drum-head presents much the same appearance as it does in suppurative disease of the middle ear. When the dermic layer is involved, vesicles filled with serum form, and voluntarily discharge their contents in eight or ten hours. At the onset of the attack there is a sharp, stinging pain, radiating to various parts of the head. In the *superficial type* it soon subsides, while in the deeper variety it may exist for some time. There is a sense of pressure, fulness, tinnitus, and moderate deafness. There may be hyperæsthesia to noises. The disease runs its course in from three to five days.

Acute myringitis—treatment: If there is marked inflammatory action, the application of the Leiter coil (Fig. 57) or the artificial leech in front of the tragus, exerts a favorable influence upon the course of the disease. The serous vesicles should be punctured, care being exercised to avoid penetrating the deeper layers. If there is an abscess in the deep layers, it should be freely incised to prevent the spontaneous discharge of pus into the middle ear. In doing this, extreme care should be used to avoid opening through the mucous layer. Should the mucous layer be incised, the pus and germs will enter the middle ear and excite a similar process there. After the puncture has been made the external meatus should be packed with a strip of sterilized gauze, which should be removed by the physician, and another one introduced in about twenty-four hours.

Chronic Myringitis.

Etiology: This form of inflammation is rare, and involves the whole surface of the drum-head. It may be due to repeated attacks of acute myringitis, especially in scrofulous and cachectic people. It sometimes follows diffused or follicular inflammation of the meatus, and is often associated with chronic middle-ear suppuration which has been followed by closure of the perforation.

Symptoms: The membrane has a moist, lustrous appearance, is grayish in color, with yellowish spots here and there upon its surface. The handle of the malleus is injected. At times the dermic layer of the drum-head is thrown off, after which the membrane beneath is more smooth, red, and velvety. In some cases small papillæ form, giving the surface of the membrane the appearance of a raspberry.

The disease is usually unattended by pain or other marked *subjective* signs. There is slight itching, and in those cases in which there is rapid exfoliation of the dermic layer, followed by a serous exudate, there is an offensive odor. This is due to the retention and decomposition of the epithelial cells and exudate.

Prognosis: The drum-head is rarely perforated. When there is a cessation of the serous exudate recovery occurs spontaneously. Whether cure is effected spontaneously or through treatment, there are a moderate thickening of the drum-head and slight impairment of hearing, although the process may have existed for several years.

Chronic myringitis—treatment: In mild cases antiseptic irrigation, followed by mopping the parts dry and dusting with boric acid powder, often causes a cessation of the secretion and restores the parts to their normal condition. In the more obstinate cases the meatus should be irrigated and a saturated alcoholic solution of boric acid instilled for ten or fifteen minutes, three times a week. When granulations or papillæ form upon the drum-head, they should be painted two or three times a week with the tincture of iodine. If this does not remove them, they should be painted with ferric chloride by means of a probe which has been dipped in the solution. The applications should be repeated at intervals of two or three days until the membrane is smooth and dry.

THE MIDDLE EAR.

Fracture of Handle of the Malleus.

It may be the **result** of (*a*) direct accidental violence; (*b*) awkward instrumentation; (*c*) blows upon the ear; (*d*) jumping from a great height; and (*e*) severe blows on the skull.

Acute and Subacute Catarrhal Inflammation of the Middle Ear.

Synonymn: Secretive Form of Middle-ear Catarrh.

Pathology: It is characterized by hyperæmia and swelling of the mucous membrane covering the walls of the middle ear, inner surface of the drum-head, and the ossicles. Hyperæmia, no doubt, also involves the mucous membrane of the antrum and mastoid cells. There is a serous exudate containing proliferated cells, a few red blood-corpuscles, and leucocytes. In the severer cases there are ecchymoses in the mucous membrane. The epithelial lining is thrown off and macerated in the serum. It usually runs its course without perforation or other serious complication. In some cases, however, adhesive bands form, binding the ossicles together and to the tympanic walls.

Etiology: It is most often an extension of a similar process from the nasopharynx by way of the Eustachian tube. It is most common in the spring and winter months, when there are sudden changes of temperature. It follows in the wake of influenza, syphilis, and the acute exanthematous fevers. Closure of the Eustachian tube from paralysis of the palatal muscles or interference with their action by the adhesion of the tonsils to the pillars of the fauces, predisposes to this affection. The presence of postnasal adenoids and postnasal catarrh also aid in its production. When the tube is closed, the air in the tympanum becomes rarefied through absorption, and this produces chronic engorgement and œdema of the lining mucous membrane. Thus the conditions are favorable for the production of the characteristic sero-mucous exudate. *The disease is common in childhood*, and is responsible for the majority of the "earaches" occurring at this period of life.

Acute and subacute catarrhal inflammation of the middle ear —symptoms: In the acute cases there is more or less *pain*, which in children is commonly referred to as "*earache*." There is a *slight febrile movement*, although it is not pronounced enough to attract attention. There is a *sense of fulness* and *pressure* in the ear, which in some cases conveys the impression of a body moving to and fro with the movements of the head. The *hearing varies* greatly at different times, thus forming a characteristic sign of this type of inflammation. In one-sided catarrhs *autophony* is present.

The *appearance of the drum-head* depends upon the amount of thickening and the quantity and quality of the secretion. If the drum-head is transparent, the upper limit of the secretion is marked by a bright line, which may be wavy, straight, double, or radiating. The color below the line is a dark yellow, while above it is a light gray. The position of the line changes with the movements of the head if the secretion is fluid; if it is thick mucus, it will change but little, if at all. The surface of the drum-head is more lustrous than normal, while the handle of the malleus is more prominent. Inflation of the tympanum sometimes imparts a peculiar appearance to the membrana tympani. That is to say, bubbles of air are seen as little rings slowly floating upward to the surface of the intratympanic fluid.

Upon examination of the *nose* and *throat* hypertrophic rhinitis, postnasal catarrh, and enlargement of the faucial and pharyngeal tonsils will often be found.

Prognosis: Mild acute cases may terminate favorably in a few days or weeks, while the subacute or chronic cases generally run a very protracted course. If the Eustachian tube is obstructed with adenoids, recovery will quickly follow their removal. If it is closed by chronic nasopharyngeal catarrh, a long course of intranasal and postnasal treatment will be required to establish their patency. Cases in which there is a copious discharge get well sooner than those in which the amount of discharge is moderate. Constitutional diseases affect the course unfavorably. In cases in which adhesive bands form a cure is problematical. Long-continued inflammations are usually associated with atrophy, and fatty and colloid degeneration of the labyrinth.

Acute and subacute catarrhal inflammation of the middle ear—treatment: This should aim at the establishment of two conditions, viz.: (*a*) the patency of the Eustachian tubes; and (*b*) the removal of the exudate.

(*a*) The *patency of the tubes* is brought about by the *correction of existing intranasal disease*, the *removal of the faucial* and *pharyngeal tonsils*, and the *reduction of the postnasal catarrh*. In cases due to paralysis of the palatal muscles, suitable treatment should be adopted.

(*b*) The *secretion* and *exudate may be removed* by tympanic inflation, or by paracentesis of the drum-head. Inflation may be done by the Politzer method or by the use of the Eustachian catheter. The tube is thus opened, air forced into the tympanum, and the fluid allowed to flow out. After inflation, the Delstanche masseur (Fig. 51) should be used to rarefy the air in the external meatus. The drum-head and malleus are thus drawn outward and made to assume their normal positions, and incipient adhesive bands are broken up. Where there is great bulging of the drum-head from the presence of a large amount of secretion, a free incision should be made at the point of greatest bulging. This affords immediate relief, although the thick mucus may not at once discharge into the meatus. Inflation will drive it into the meatus, or it may be drawn there with the Delstanche masseur. If the mucus is very tenacious, it may be removed with forceps, or softened with instillations of an aqueous solution of sodium bicarbonate.

In the *early acute stage*, the application of the artificial leech in front of the tragus will remove the congestion and relieve the pain. Warm water or oil dropped into the meatus will also relieve it. I have often used a few drops of chloroform upon a piece of cotton in the bowl of a clay pipe, placing the bowl to my mouth, and blowing the fumes through the stem into the ear. *Politzerization* is, however, the most rational method of relieving earache in children, as it overcomes the vascular tension causing it.

Adhesions, Thickening, and Retractions Following Chronic Exudative Catarrhal Inflammation of the Middle Ear.

Fibrous bands are left as a result of chronic inflammation.

Effect on membrane: They may bind the membrane to the inner wall of the tympanic cavity or to the incus and stapes. When the drum-head thus becomes adherent, exhausting the air in the external auditory meatus with the Delstanche masseur and the Siegle otoscope (Fig. 51) does not cause an outward movement of the entire drum-head. The adherent portions remain fixed, while the rest of the membrane moves outward.

The bands may also extend from the ossicles to the inner wall of the tympanum. All these conditions are attended by more or less deafness and tinnitus.

Effect on tensor tympani: The fibrous formations and the subsequent retraction sometimes cause the tendon of the tensor tympani muscle to be shortened. The focussing and conduction functions of the drum-head and ossicles are thus impaired.

Effect on cavity of the tympanum: This is often very much diminished in size by the chronic hyperæmia, hypertrophy, and hyperplasia of the mucous membrane.

Dry Catarrh, or Sclerosis, of the Middle Ear.

Synonyms: Adhesive Processes in the Middle Ear; Proliferous Inflammation of the Middle Ear; Chronic Catarrh of the Middle Ear.

Etiology: The adhesive process may follow a subacute exudative process or it may appear independently.

Deafness indicating an *insidious advancement* of the dry catarrhal process is more likely to be associated with *labyrinthine disease* than is deafness due to exudative catarrh.

Sometimes the labyrinthine disease predominates or seems to be primary, so that it is possible that both are due to trophic changes now little understood.

The process may also result from (*a*) frequent attacks of acute catarrh; (*b*) nasopharyngeal disease, which prolongs the ear catarrh; (*c*) paralysis and degeneration of the tubal muscles; (*d*) hereditary predisposition (about 40 per cent.); (*e*) general constitutional diseases and dyscrasiæ; (*f*) living in damp houses; (*g*) sea-bathing and diving; and (*h*) the excessive use of alcoholics. The disease is common in middle and old age.

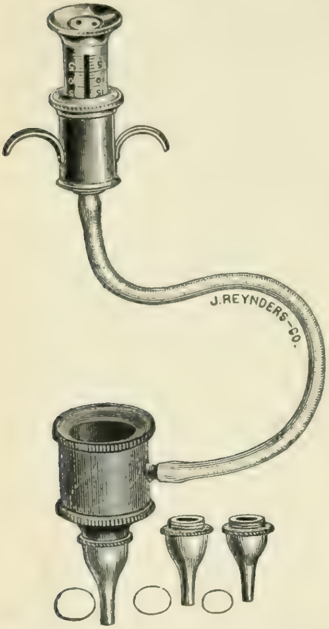
Pathology: (*a*) The *first stage:* There is a hyperæmic boggy, succulent, yellowish-red membrane with excessive round-cell infiltration. (*b*) *Second stage:* The membrane is smooth, thick, and of a pale tendinous gray color. It is firmly adherent to the parts beneath, the round-cell infiltration having become fibrous connective tissue. (*c*) *Third stage:* The fibrous bands cross in various directions, binding the ossicles together and to the walls of the tympanum.

There may also be ankylosis at the foot-plate of the stapes from fibrous thickening or calcification about the margin; the fenestra rotunda may be affected by fibrous thickening and calcification; sometimes the Eustachian tube is closed by thickening of the mucosa, or by fibrous stricture, the cartilaginous part of the tube being most commonly affected; the palatine muscles at the orifices of the tubes sometimes undergo fatty degeneration and cicatricial shortening; the intratympanic muscles may also be impaired by atrophy, fatty degeneration, and cicatricial or colloid degeneration.

Dry catarrh of the middle ear—symptoms: *The appearance of the membrana tympani* is dull and opaque, or it may show opacities about its border. There may be calcareous deposits, although they are rare. The handle of the malleus is less distinctly visible than normal, and the cone of light is irregular or lost. The drum-head is retracted, the handle of the malleus foreshortened, and the processus brevis more prominent. There may be circumscribed retractions of the drum-head from adhesions and cicatricial bands. In advanced sclerotic cases the external meatus may be free from cerumen, the skin being white and glistening.

Subjective symptoms: The tinnitus is often very annoying and continuous. It is worse in bad weather, after fatigue, mental exertion, or excessive drinking. The patient complains of fulness and giddiness, aprosexia, and hearing for speech is often very dull, while the drop of a needle may be distinctly heard. Paracusis Willisii, or improved hearing in the presence of noises, is a common and characteristic symptom of this form of deafness. There is a gradual increase in the dulness of hearing, although there may be intermissions during which no change occurs. Complete deafness is rare. Bone conduction is usually increased, although it may be decreased when the labyrinth is diseased at the same time, and when the fibrous contractions force the foot-plate of the stapes into the oval window, thereby increasing the intralabyrinthine pressure.

FIG. 51.



Siegle otoscope, with masseur of Delstanche attached.

The **prognosis** of dry catarrh is better in those cases following the secretive form of catarrh than it is in the insidious type, which is so often complicated with labyrinthine disease. It is unfavorable in proportion to the degree and persistence of the subjective noises. Those cases which improve after inflation are favorable. If the deafness develops rapidly, it is bad. With a permeable Eustachian tube, unceasing noises, and progressive deafness, the prognosis is very unfavorable. If, after inflation, bone conduction remains poor, labyrinthine disease is probably present. This type of deafness is usually incurable when found in old and scrofulous people.

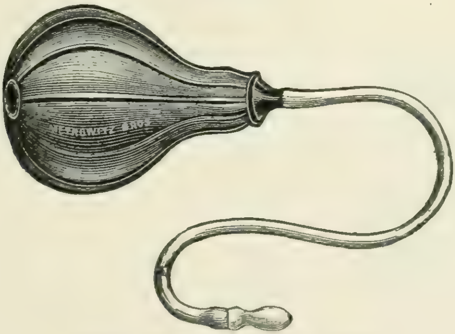
Dry Catarrh of the Middle Ear—Treatment.

Non-operative: In nearly all cases treatment is unattended by favorable results. Indeed, it is sometimes followed by very bad results. Pneumatic massage, applied through the external meatus, together with inflation of the middle ear, is the best form of treatment in most cases. Pneumatic massage consists in the alternate rarefaction and condensation of the air in the external meatus with the Delstanche masseur (Fig. 51); or with a pump attached to an electro-motor engine.

In the spring of 1893 the author attached the Siegle otoscope to the pneumatic hammer attachment of a dental electric engine and thus applied pneumatic massage with electro-motor power. Since then a small electro-motor engine has been devised for this especial purpose. The treatment should be given for five to ten minutes three times a week; and should not be given for a period of more than six weeks, as atrophy of the drum-head may follow a prolonged course of treatment.

Inflation may be done with the Politzer bag (Fig. 52) or the catheter (Fig. 53). The injection of ℳvj of 2 per cent. pilocarpin solution, oils, and vapors through the catheter into the middle ear is sometimes followed by improvement in hearing. The improvement, however, is perhaps almost altogether due to the distention of the middle-ear cavity, rather than to the effect of the drug itself. Constrictions of the Eustachian tube may be overcome by the use of bougies made of whalebone, celluloid, or catgut. A bougie provided with a metal

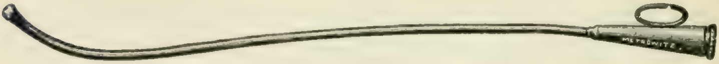
FIG. 52.



Politzer bag, with glass nose-piece.

tip and an insulated wire shank may be introduced through the Eustachian catheter to the point of constriction, and a current of about 5 ma. of electricity turned on, the constrictions being thus reduced by electrolysis.

FIG. 53.

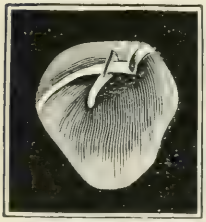


Blake's Eustachian catheter.

Operative treatment: (*a*) *Artificial perforation* of the membrana tympani is often attended by improved hearing and lessened tinnitus. It is best performed with the electric cautery. The opening remains, however, for only a short time. Many ingenious methods have been devised to render the opening permanent, but they have been attended with little success.

(*b*) *Plycotomy*, or section of the posterior fold (Fig. 54), of the membrana tympani, is done in cases of great deafness with subjective noises and pronounced retraction of the handle of the malleus. The section is made immediately behind the short process, or midway between the process and the peripheral end of the fold, cutting from above downward. The handle of the malleus is thus allowed greater motion, the subjective noises are diminished, and there is improvement of hearing lasting for a few weeks or months.

FIG. 54.



Plycotomy: handle of malleus foreshortened; anterior and posterior folds of the drum-head very prominent. (Bacon.)

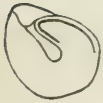
(*c*) The *section* of the *anterior ligament* of the malleus is sometimes followed by improvement of hearing and subjective noises.

(*d*) *Tenotomy* of the tendon of the tensor tympani and stapedius muscles is also performed for the same purpose.

(*e*) *Ossiculectomy:* If there is ankylosis of the malleus and incus, or firm adhesive bands interfering with their mobility, the bones may be extracted through an incision in the drum-head. This may be done by one of two methods (Figs. 55

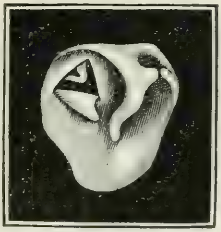
and 56). This operation was once quite popular, as it promised great relief in this type of deafness. A larger experience has demonstrated that the beneficial results in most instances are of but temporary duration. Occasionally, however, it is followed by marked permanent improvement. (*f*) In very rare cases there is ankylosis of the foot-plate of the stapes, and *mobilization* may be attempted. This is a very difficult and delicate operation, and should only be attempted by a trained and skilful operator.

FIG. 55.



Blake's incision in posterior-superior quadrant. (Bacon.)

FIG. 56.



Triangular incision in the posterior-superior quadrant for exposing the articulation of the incus and stapes. (Bacon.)

Suppurative Otitis Media.

Varieties and course: Suppurative inflammation within the cavity of the middle ear may occur in either the acute or the chronic form. The *acute type* may be either primary or an acute exacerbation of the chronic form. The primary acute type is not so dangerous as the chronic or the acute exacerbation occurring during the progress of the chronic form.

Suppurative otitis media is characterized by a primary acute suppurative inflammation which may subside, leaving healthy tympanum; or it may continue indefinitely in a chronic form with acute exacerbations. The drum-head is usually perforated, the discharge taking place chiefly through the external auditory meatus, although there may be more or less discharge through the Eustachian tube into the nasal pharynx. The *primary acute type* and the *acute exacerba-*

tions of the chronic form are attended by marked febrile symptoms and more or less pain in the ear and mastoid.

Suppurative otitis media—etiology: (*a*) The specific or infectious fevers in which the peculiar pyogenic organism incident to them gain access to the tympanum through the Eustachian tube. (*b*) Catarrhal states of the nasopharynx are predisposing causes. (*c*) The presence of enlarged pharyngeal tonsils (adenoids) is often the cause of middle-ear suppuration, as they close the Eustachian orifice or interfere with action of the palatine muscles. (*d*) Exposure to inclement weather, diving, and injuries by direct or indirect violence may cause it. (*e*) Influenza is especially liable to excite a very destructive type of middle-ear inflammation.

Diphtheria and scarlet fever are also often followed by severe suppurative otitis media with great destruction of the intratympanic tissues and the drum-head.

The *microörganisms* commonly found in the discharge are the Streptococcus pyogenes, the Staphylococcus pyogenes albus and aureus, Friedländer's pneumobacillus, Fränkel's pneumococcus, and the tubercle bacillus.

Pathology: In *primary acute otitis media* there is rarely an extension of the pathologic process to the cranial cavity. This is due to the fact that the mucous membrane and bony walls of the middle ear remain intact, and thus obstruct the progress of the infective matter inward. If the inflammation continues for two years or more, the bloodvessels of the mucosa may become thrombosed and cause degenerative changes in it, and thus give rise to an extension of the pathologic process to the meninges and brain. If the thrombi are infected, they may become the source of infective emboli and be carried to the brain, spleen, liver, kidneys, and lungs. Meningitis, pneumonia, etc., may follow as direct results. It is now a pretty well recognized fact that most cases of meningitis not due to tuberculosis are directly traceable to chronic suppurative otitis media. It must not be forgotten, however, that suppurative disease of the accessory sinuses of the nose is also responsible for a certain number of cases.

Granulation-tissue springs up at the points of ulceration and bone-necrosis in an attempt to repair the damage done to the tissues. They cause absorption of the contiguous bone by pressure-necrosis at the same time. Their presence also obstructs the flow of pus and causes its retention. This leads to septic infection and the pressure-symptoms so commonly observed in mastoiditis.

If the aqueductus Fallopii (through which the facial nerve passes) is exposed by the necrotic process, the infection may gain entrance to the cranial cavity through the sheath of the facial nerve.

Aural polypi are simply masses of granulation-tissue, and usually signify necrosis of the bony walls of the tympanic cavity. The roof of the attic is the point usually involved, although any of the walls may be thus affected.

General Prognosis of Suppurative Middle-ear Disease.

The **primary acute type** usually runs a short course. Hence is not likely to extend into the cranial cavity; but it may become chronic.

Facial paralysis sometimes occurs in the course of the chronic form, and may be due to necrosis of the posterior-superior wall of the tympanum. It is a serious sign, as a radical mastoid operation (Schwartz-Stacke) may be required.

Recurrent attacks of pain in the mastoid are signs of chronic mastoiditis.

A fetid odor is also a sign of chronic mastoiditis, and requires careful examination. It may not be noticeable until the middle-ear cavity is searched with a probe, when it becomes quite pronounced and offensive.

Perforation of the membrana tensa in the posterior-superior quadrant is significant of caries of the body of the incus, and may require the removal of said bone before a cure can be effected.

Perforation of the membrana flaccida (Shrapnell's membrane) is a sign of necrosis of the malleus or incus, or of both bones. The attic walls are also sometimes involved, in which case the danger may be great.

Perforation elsewhere in the drum-head signifies a pathologic process of much less gravity, one probably limited to the middle ear proper. Such cases afford a much more favorable prospect of cure by simple local remedies than perforation in the posterior-superior quadrant or in Shrapnell's membrane.

A marked diathetic state renders the prospect of cure under either topical treatment or surgical measures very problematical. Iron, cod-liver oil, nutritious food, pure air, and sunshine are especially indicated.

A small amount of discharge is usually regarded lightly by both patient and physician. Personally, the author does not hold such a view. Thrombosis of the vessels of the mucosa is most likely to occur under such conditions. Suddenly acute mastoid or middle-ear symptoms develop, and the gravity of the case becomes apparent. Too much stress cannot be laid upon this point. Meningitis occurring in the course of a so-called mild chronic suppurative otitis media is usually regarded as idiopathic, whereas, in fact, it may be but an extension of the infection from the middle ear.

The Symptoms of Suppurative Otitis Media.

These vary according to the acuteness or chronicity of the disease and the amount of obstruction to the drainage of the middle ear.

In the **acute stage** or **acute exacerbation** there may be pain of an excruciating character. It may be located in the ear or in the mastoid bone, according to the parts chiefly involved. If the mastoid cells are affected, pressure over the mastoid will be attended by pain. The auricle is usually somewhat swollen and tender.

The acute manifestations are attended by fever, while the chronic form is but slightly or not at all thus affected.

The **chronic form** is characterized by little or no pain unless an acute exacerbation occurs. If the drainage from the mastoid cells becomes obstructed, pain, swelling, redness, and tenderness quickly develop. The condition commonly known as "acute mastoiditis" is present. In such cases the patient will usually tell the physician that there had been profuse discharge from the ear, which suddenly stopped when the pain began.

Whether the inflammation be of the **acute** or **chronic** type, subjective noises, impaired hearing, giddiness, and loss of the sense of taste and smell are usually present.

Suppurative Otitis Media—Seen on Examination.

First stage: The dermic layer of the drum-head is injected at its periphery, over the membrana flaccida and along the handle of the malleus. As the exudate forms, the drum-head becomes bulged outward. The handle of the malleus is no longer visible. If the attic is chiefly involved, the bulging will be in the membrana flaccida. The color varies from a red, sodden appearance to a greenish or purplish-gray. The skin covering the mastoid and auricle may be tender and slightly œdematous. Febrile symptoms are present.

Second stage: After a few days the process as described in the first stage ends by resolution (rare); or by perforation of the drum-head, which may be located in the membrana flaccida or in the pars tensa, according to the chief seat of the inflammation. It may be high, low, posteriorly, anteriorly, or in the centre of the pars tensa. It may be round, oval, kidney-shaped, or irregular. It may vary in size from a pin-point to that of the entire drum-head. The secretion may be slight or excessive, thin and admixed with serum, or rich in creamy pus. In some cases it is mixed with blood and gritty material. Cholesteatomatous matter may also be discharged. Odor may be present, an important symptom in cases of retention of the secretion or of bone-necrosis.

The external meatus after a time is irritated and excoriated from the discharge. Polypi may project through the perforation and completely obscure the drum-head. If the perforation is large, the inner wall of the tympanum may be seen. The mucous membrane will appear swollen and red. It may be smooth or granular, or it may be obscured by large granulations. The ossicles are sometimes seen.

The lymphatic glands below the ear are usually enlarged and tender. They may become tubercular.

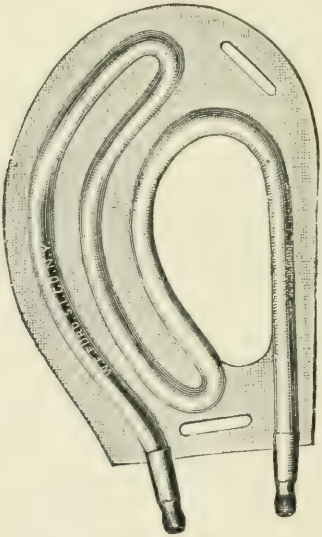
Third stage: This is the process following the cessation of active secretion. The drum-head is lustrous and hyperæmic about the circumference, with vascular injection centring about the umbo. Later, it is of a dull grayish color, usually with perforation. It may be retracted and adherent to the inner tympanic wall. The handle of the malleus is often retracted and foreshortened, while the short process is quite prominent.

Chronic form: This is the *third stage*, lasting over a few weeks. Otorrhœa is intermittent, with or without odor. It may diminish until belief in a cure is established; but after several years an acute mastoiditis may develop, or meningitis or brain abscess, due entirely to the supposedly healed ear disease.

Suppurative Otitis Media—Treatment.

First stage: Pain and congestion may be relieved by antiphlogistic measures. Local heat and cold and the abstraction of blood are the most reliable remedies for this purpose. Dry cold, in the form of crushed ice in a rubber bag or ice-water flowing through a Leiter coil (Fig. 57), is usually more efficacious than dry heat. A layer of flannel should be placed between the skin and the bag or coil, to prevent unpleasant symptoms. Artificial leeching in front of the tragus or over

Fig. 57.



Leiter coil.

the mastoid process should be done when there are pronounced inflammatory signs which will not yield to applications of heat or cold. In case the pain does not abate, the drum-head should be freely lanced whether it is bulging or not. If it is bulging, it should be lanced even if pain is absent.

Medicinal applications in the meatus are often useful. Warm solutions of two grains of morphine to an ounce of water; 5 per cent. of carbolic acid in glycerin; and warm olive oil sometimes afford great relief from pain. It may be necessary, however, to administer opiates when the pain is quite severe and prolonged.

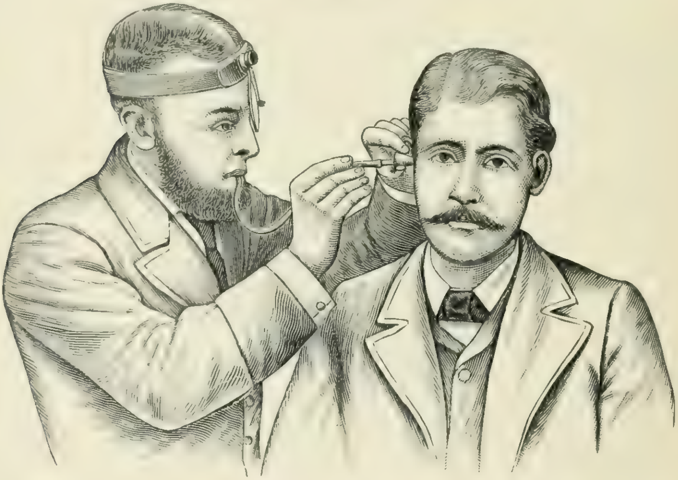
The nose and nasopharynx should be treated with warm alkaline sprays and the application of a 10 per cent. oily solution of menthol. The menthol will relieve the congestion of the mucous membrane about the Eustachian orifices and thus promote the patency of the tubes.

Should acute *mastoid symptoms* become prominent and persistent, it may be necessary to remove the morbid material from the middle ear, attic, antrum, and mastoid cells (see Mastoid Operation).

Second stage: After spontaneous or artificial perforation, the external meatus and middle ear should be thoroughly cleansed with warm boric-acid solution. A 50 per cent. solution of 15-volume hydrozone should then be instilled into the meatus and Politzerization performed. Pus is thus driven from the middle ear into the meatus, the hydrozone entering the tympanum, where it destroys the remaining portion of pus. In doing this the patient should incline the head to the opposite side, so that the solution will remain in the meatus during inflation. The ear should be syringed again with warm boric-acid solution and dried with a cotton-wound applicator. The middle ear should be still further dried by instilling a 50 to 95 per cent. solution of alcohol into the meatus. The alcohol by its hygroscopic action takes up the remaining moisture and causes the small bloodvessels of the swollen and glandular membranes to contract. It not only takes up the water left in the middle ear, but also abstracts more or less of the serous contents of the mucous membrane itself. The meatus should then be packed with sterilized gauze, which acts as a protective dressing and keeps the middle-ear cavity comparatively dry by capillary attraction. The treatment should be repeated every twelve to forty-eight hours, according to the amount of discharge, and should be done by the physician himself, as it is practically impossible to instruct the patient or members of the family to carry it out properly.

Finely powdered boric acid may be used instead of the gauze in cases with a copious yellow pus secretion unattended by pain or other pressure-symptoms. It should be blown (Fig. 58) into the meatus until it is about half-filled, and a cotton dressing inserted over it. It should be renewed as often as the liquid discharge softens and dissolves it. In small perforations it is not so useful, as there is not enough moisture to dissolve it, but just enough to cause it to form a solid mass which obstructs the flow of pus. In such cases it may do more mischief than good.

FIG. 58.



Insufflation of powders into the ear under illumination by the forehead mirror.

Saturated solutions of boric acid or iodoform in alcohol justly hold a high position as remedial agents. The astringent and antiseptic action of these solutions, together with their power to abstract moisture, make them ideal remedies to check the development of pyogenic bacteria and the formation of granulation-tissue.

In the more *chronic* stage of the disease the instillation of a 95 per cent. solution of alcohol (commercial alcohol) into the ear for ten or fifteen minutes every twenty-four to forty-eight hours is often followed by very marked improvement.

The dry gauze treatment: Simple mopping with a cotton-wound applicator, followed by a dressing of plain dry gauze, packed from the bottom of the meatus or middle-ear cavity, is in many cases the very best kind of treatment. A strip of gauze three-eighths to one-half inch wide, and ten inches long, should be used for this purpose. It should be replaced as often as the deeper end becomes moistened. It absorbs the pus and serum, and thus prevents the formation of a culture-medium for bacteria. Herein lies the great value of this method of treatment.

Ossiculectomy: The malleus and incus are often more or less denuded of mucous membrane and periosteum; or they obstruct the flow of pus from the attic and antrum in suppurative disease of the accessory cavities of the ear. When either of these conditions exist they should be removed. The handle of the malleus should be dissected from the drum-head and inflammatory fibrous tissue in which it may be imbedded. The ligaments attaching it to the attic walls should also be severed. It is freed by incising the drum-head, as shown in Figs. 55 and 56.

The whole drum-head may be removed by extending the incision around its periphery, thereby affording a clear view of the middle ear. The ligamentous attachments should be severed with suitable curved knives (Sexton's middle-ear set). The handle of the malleus should be seized near the head of the ossicle with a pair of small forceps and pulled downward and outward with a rocking motion. The stapes sometimes comes away at the same time. If it does not, it should be removed with delicate ear-forceps. As the incudostapedial articulation is usually a very frail one, special operative measures to separate it are not necessary. The "floor" of the attic being thus removed, the flow of pus from the antrum and mastoid cells and attic into the middle ear is unobstructed unless the disease-process has given rise to granulations or other morbid material in the antrum and mastoid cells; in which case the obstruction may be farther back than the "floor" of the attic. Simple ossiculectomy may not be followed by the relief hoped for. A radical mastoid operation may then be required.

The Mastoid Operation.

The **various steps** of the mastoid operation are: (*a*) preparation of the patient; (*b*) the postauricular incision; (*c*) the anatomical landmarks; (*d*) exposure of the antrum; (*e*) exploration through the exposed antrum; (*f*) the removal of the bony wall between the antrum and middle ear; (*g*) the removal of the morbid material from the middle ear and the mastoid cells; (*h*) how to avoid injury to the facial nerve, horizontal semicircular canal, and fenestra ovalis; (*i*) closure of the wound; (*j*) postoperative treatment; and (*k*) the results of the operation.

The above is for *chronic cases* and their *acute exacerbations.* For *primary acute mastoiditis* the operation may be shortened by simply entering the antrum and mastoid cells, and it may not be necessary to remove the postsuperior wall of the meatus or the ossicles.

Preparation of the patient: The operation should be done in a hospital if possible, as it is a prolonged one, and may require every advantage for its successful performance. A superficial operation will suffice perhaps to avert immediate danger, but it will not cure the patient of the chronic pyogenic disease, which may at any subsequent time place his life in jeopardy. Do a thorough operation once for all, and cure both the acute (immediate source of danger) manifestation and the existing chronic condition (pending source of danger).

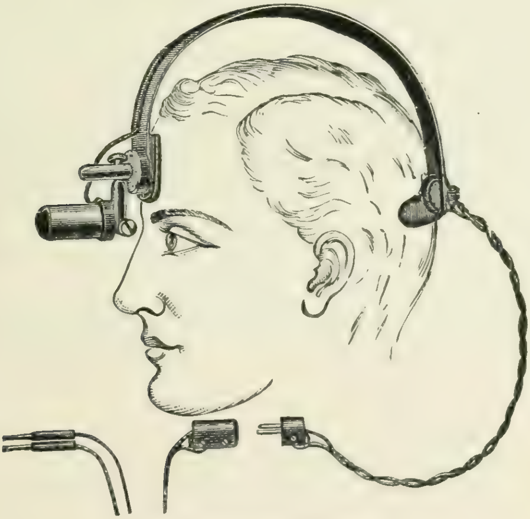
Shave the head for two inches around the auricle, twenty-four to forty-eight hours prior to the operation. Scrub the skin with green soap and boiled water, after which it should be washed with alcohol. The scalp should then be washed with spirit of turpentine, to remove the sebaceous matter. The external meatus should also be thoroughly irrigated and scrubbed. Pack the meatus and cover the whole side of the head with moist carbolized (or sterilized) gauze. These should not be removed until the patient is under the influence of the anæsthetic.

The patient should be placed upon a light diet, and be given a cathartic on the evening previous. He should take no food within twelve hours of the operation.

After anæsthesia is well established remove the dressings and scrub the parts with soap and water followed with an alcohol bath. Pin a sterilized towel or rubber cap around the patient's head and place it on a cushion, to overcome the shock of the blows of the mallet.

The wound should be well lighted during the operation. This may be done with a lamp and head-mirror. My custom is to use an electric head-lamp (Fig. 59) attached to a powerful storage battery.

FIG. 59.



Direct illumination of the ear.

The postauricular incision: This should begin at the tip of the mastoid process, and follow, within one-quarter inch from the line of union between the auricle and the skin over the mastoid, to a point immediately over the superior auricular attachment. The tip of the mastoid should be located and the scalpel inserted through the skin and periosteum, cutting both as it follows the auricular attachment. The periosteum should be elevated or loosened with the periosteal elevator and the bleeding points seized with artery-forceps. The periosteum and skin of the postsuperior wall of the meatus should also be separated from the bone and cartilage with a small elevator, after which the skin meatus should be severed with a curved knife (tip curved on the flat) as close to the drumhead as possible. The ear is pulled forward and the posterior edge of the wound backward and held with retractors in the hands of assistants. A self-retaining double retractor is a better arrangement, as it leaves the assistant free to do other things.

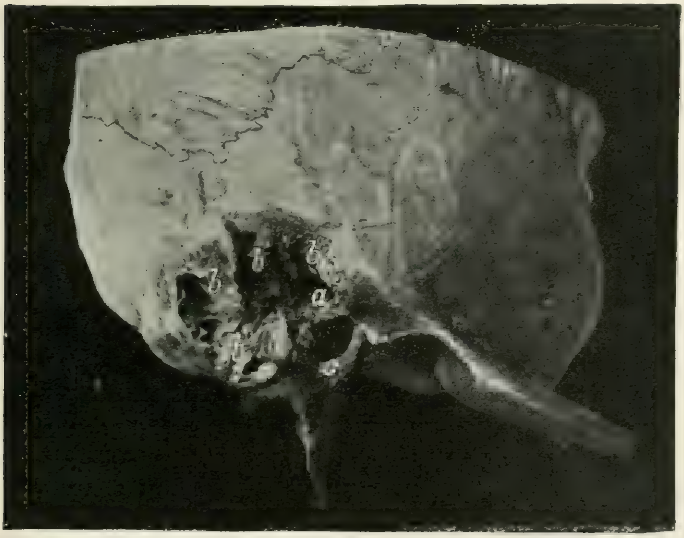
The anatomical landmarks: Having exposed the bone and meatus, certain landmarks should be sought as guides to the antrum by such a route as will avoid injury to the lateral sinus, the middle fossa of the cranium, and the facial nerve.

By operating within the limits of the *suprameatal triangle*, and following the direction of the external auditory meatus to a depth about three-eighths of an inch less than the depth of the bony meatus and middle ear combined, the sinus, cranial fossa, and facial nerve will not be injured, except rarely. The boundaries of the suprameatal triangle are as follows: (*a*) the antero-superior boundary is formed by the posterior ridge or root of the zygoma; (*b*) the posterior boundary is an imaginary line extending from the posterior end of the zygomatic root, downward and slightly forward to the posterior tubercle of the external auditory process; (*c*) and the antero-inferior boundary is the curved margin of the postsuperior wall of the meatus.

An opening extending from the centre of the triangle inward in a direction parallel with the postsuperior wall of the external auditory meatus will almost invariably expose the cavity of the antrum, unless it has been obliterated by sclerosis or other process. Herein is the reason for measuring the depth of the meatus and tympanum before beginning the removal of bony structure.

Exposure of the antrum: By knowing the depth of the parts named and subtracting three-eighths of an inch (the outer wall of the antral cavity is about three-eighths of an inch nearer the surface than the inner wall of the middle-ear cavity) the operator knows at what depth he should enter the antrum. And he also knows that if he does not enter the antrum at that depth it is obliterated (rare); or that it is a little deeper; or that he has misjudged the direction. Careful exploration with a curved dental canal-probe should be made into every small opening presented on the walls of the bony cavity. If one is found extending forward toward the middle ear, it should be cautiously followed. If it goes forward into the middle ear, it is probably the antrum. If it does not, it is probably a mastoid cell, and the operation should be directed still deeper in search of the antrum. Unless certain of the landmarks, it is well to go very slowly, as there is a liability of inflicting injury to the facial nerve or horizontal semicircular canal. Having located the antrum, its entire external wall

FIG. 60.

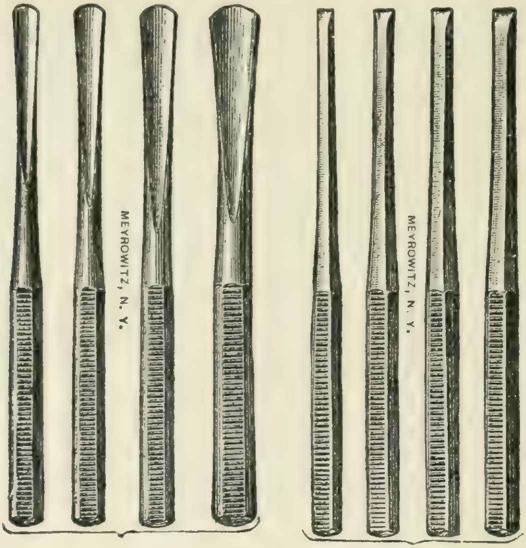


Section showing the antrum situated just above and a little behind the external auditory canal and communicating with the mastoid cells. (Bacon.)

should be removed, so as to expose it to inspection and operation.

In many *acute mastoid* cases these steps will be greatly modified by the presence of carious and necrotic bone. Where the bone is softened the operator should follow the track of carious bone to any part or in any direction. But the location of the antrum and the facial nerve should not be lost sight of. The operation should aim at a complete exposure of the antrum (Fig. 60), no matter where the carious bone may be found. The disease in all probability started in the middle ear, hence this cavity should also be opened for thorough inspection. The ultimate and permanent cure will often depend upon the establishment of free drainage through the middle ear and external meatus. Hence the importance of exposing the antrum so that the adaditus may be enlarged if necessary.

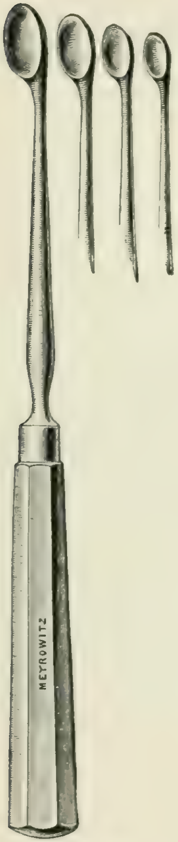
FIG. 61.


MEYROWITZ, N. Y.
MEYROWITZ, N. Y.

Chisels and gouges. (Schwartze.)

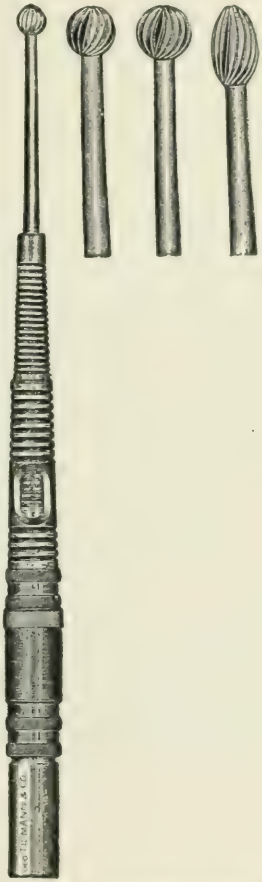
Chisels and gouges varying in width from three-sixteenths to three-eighths of an inch, the dental engine and burrs (Figs. 61 and 63) of suitable size, or the Russian perforator may be used to open the antrum. The chisel is the best instrument in the hands of the occasional operator, while the dental burr is perhaps the safest and best instrument in the hands of

FIG. 62.



Sharp spoons.

FIG. 63.

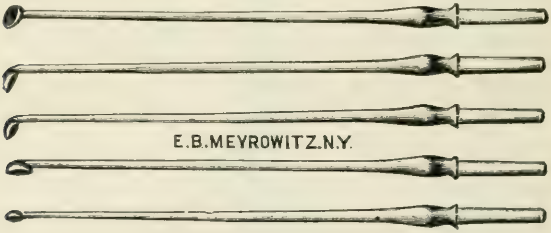


Macewen's burrs.

one using it frequently. With it the wound is always smooth and easily explored. The Russian perforator is of special value in operating on chronic cases in which the mastoid is thickened and sclerosed. It is not a suitable instrument where there is a softened carious mastoid, the chisel and spoon being best adapted to these cases. The presence of a small canal or cell filled with granulations or cholesteatoma is more easily seen and explored when either the burr or Russian perforator is used, as they leave a smooth surface. The use of strong illumination becomes imperative when the object of the operation is not only to relieve the immediate danger, but to remove the source of future infection at the same time. To do less than this is culpable negligence.

Exploration through the exposed antrum: After having removed the outer wall of the *antrum* the *cavity* should be thoroughly explored by inspection and with a small probe for caries and necrosis. The presence of granulation-tissue at any point is presumptive evidence of denuded bone. The granulations should be carefully removed and the base from which they spring probed for soft or roughened bone. This, if found is best removed with a sharp spoon-curette (Fig. 62). The large curettes usually used for this purpose are only adapted to large softened areas. The *smaller foci* should be scraped with smaller spoons (Fig. 64). The dental burr may be used for this purpose. Extreme care is necessary in working in the region of the facial nerve, which lies just under the bridge of bone left between the meatus and the operative opening made into the antrum. It may, however, be on the inner floor

Fig. 64.

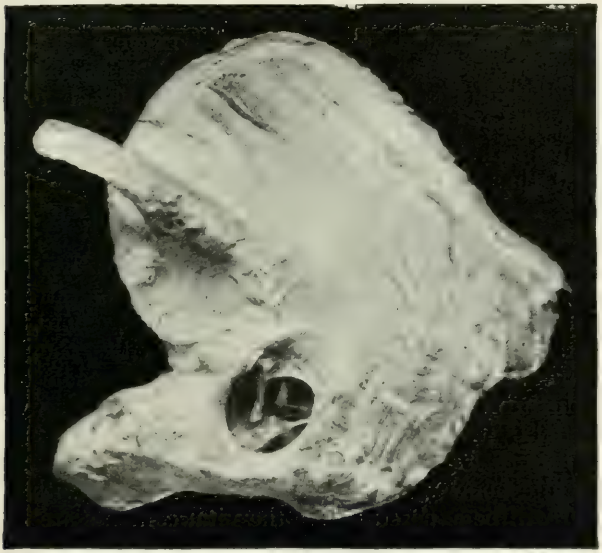


Politzer's sharp spoons.

of the antrum near the bridge of bone, especially that portion of the bridge below (erect position) the adaditus. The adaditus is the opening connecting the antrum with the attic ("aditus," meaning the attic). Carious bone in this region should be carefully curetted, the muscles of the face being watched for twitching movements.

The *mastoid cells* drain into the antrum, and they should be inspected and opened if necessary. If granulations, carious bone- or cholesteatomatous material is found in them, they should be freely opened with the chisel or burr, and all morbid material removed.

Fig. 65.



Drum-head almost entirely destroyed. Rim of membrane left in the upper portion. Ossicles bound down by adhesions. Long handle of malleus attached to inner wall of middle ear. (Bacon.)

The *middle-ear cavity* is next submitted to inspection and exploration with the probe. The ossicles and tympanic walls should be tested for bare bone and adhesive bands (Fig. 65). If either is found seriously involving the ossicles, they should be removed at a later period in the operation. If the walls of the tympanum are carious, they should be curetted. This cannot be thoroughly done with the bony wall standing between the antrum and the external meatus. Its removal is the next step in the operation.

The removal of bony wall between the antrum and middle ear: This is best accomplished with chisels or the dental burr. Bone-forceps may be used to advantage in the superficial portion, but are not adapted to removal of the deeper portion on account of the proximity to the facial nerve and horizontal semicircular canal. In the deeper portion only the *upper part* (erect position) of the wall should be removed. The *lower portion* is usually over the facial nerve. If this precaution is observed, the bugbear of the operation is avoided, namely, the injury of the facial nerve. If chisels are used, they should be very sharp, to enable the operator to remove the bone in thin shavings. If they are dull, the bone will chip and thus inflict injury to the nerve.

The removal of morbid material from the middle ear and mastoid cells: The middle-ear cavity being fully exposed to view, the *malleus and incus* should be carefully inspected for pathologic changes, and if their function is destroyed by adhesions or necrosis, or they form an obstruction to the free drainage of the middle-ear and accessory cavities, they should be removed. The *stapes* should not be disturbed, as its removal would disturb the intralabyrinthine pressure. This would be manifested by the Ménière group of symptoms.

The *facial nerve* and semicircular canals are surrounded by dense white bone, which upon careful dissection with a burr or chisel will be exposed before the vital contents are injured. Hence the necessity of exercising extreme caution at this stage of the operation. The location of the nerve is sometimes apparent on the inner wall of the antrum, near the *adaditus*, as a rounded ridge, slightly elevated above the surface.

The removal of the morbid material should be most thoroughly done, in order to prevent the continuance of the suppurative and carious processes subsequent to the operation. The most painstaking search for carious, necrotic, and granulation material should be made in every case.

How to avoid injury to the lateral sinus, facial nerve, hori-

zontal semicircular canal, and fenestra ovalis: A line drawn from the parieto-squamo-mastoid junction to the tip of the mastoid indicates the position of the *sigmoid sinus*. The knee of the sigmoid sinus often extends much nearer the external meatus than this line indicates. If, however, in opening the antrum the chiselling or drilling is confined to the limits of the suprameatal triangle and directed parallel with the post-superior wall of the meatus, the lateral sinus will not often be injured. Proximity to the sinus is indicated by the presence of dense white bone. If the chiselling is carefully and cautiously done, the operator will know by this sign that he is very near it. In size the sigmoid may be compared to the side of the index-finger.

A knowledge of the location of the facial nerve and horizontal semicircular canal, together with the surrounding investment of dense white bony tissue, will enable the operator to avoid injuring them. Only the upper portion (erect position) of the wall between the antrum and the middle ear should be removed, as the *facial nerve* is easily injured beneath the lower part. The *horizontal semicircular* canal passes near the curve of the facial nerve, on a little deeper plane, and is therefore not so liable to injury. The facial nerve also passes beneath the superior portion of the wall between the meatus and antrum, but as it passes beneath the inner wall of the adaditus is not so liable to injury during the operation.

The *oval window* need not be injured if the inner wall of the tympanum is respected during curettement of the middle ear.

Closure of the wound: Having removed all morbid material from the middle ear, antrum, and mastoid cells, and having established conditions favorable for the free drainage of the same, the external wound should be partially or completely closed. Irrigate the cavity with warm boric-acid solution, the head hanging downward over the end of the table, to prevent entrance of fluid into the trachea through the Eustachian tube and pharynx. (Irrigation may be dispensed with and dry sterilized dressings applied instead.) All particles of bone and morbid material being thus washed away, the wound should be dried, partially filled with a powder composed of one part of iodoform and four parts of boric acid, and packed with gauze moistened with the compound tincture of benzoin. The external meatus should be treated likewise, the gauze being firmly packed to hold the meatus in its normal shape and position during the healing process. If marked granulation-tissue or virulent pus infection is present, the cavity should be mopped with the tincture of iodine to reduce fragments of granulation-tissue that may have escaped notice and to stimulate healthy granulations. The upper portion of the skin-wound is sutured with silk, horsehair, or catgut, while the most dependent portion, or that portion over the excavation in the bone, is left open for drainage and the introduction of subsequent dressings. A pad composed of several layers of gauze is placed over the ear and mastoid process. A liberal quantity of cotton should be placed over this, and a suitable bandage applied to hold them in position.

Postoperative treatment: The first dressing should not be removed for six or eight days, unless there are positive signs of local inflammatory reaction. The antiseptic powder and the compound tincture of benzoin will prevent infection and decomposition, so that when the dressing is removed the wound will be clean and free from odor. The sutures may be removed at this time. Subsequent dressings should be applied every other day. If freshly sterilized gauze is used, it will be unnecessary in most instances to irrigate the wound. The dressing should be applied loosely in the middle ear and antrum, through the postauricular opening, and through the external meatus. The external wound should be allowed to heal by granulation, as in this way the formation of non-vascular connective tissue is favored.

The results of the operation: The objects of the operation are: (*a*) To avert impending danger from the progress of caries and necrosis in the course of secondary acute and chronic mastoid disease. In secondary acute mastoiditis the bony tissues undergo very rapid degeneration; while in the chronic form the process is a much slower one, oftentimes giving rise to no marked symptoms. (*b*) Another object of the operation is to establish permanent free drainage of the mastoid cells, antrum, and middle ear. (*c*) Dense non-vascular fibrous tissues forms in the place of the bony tissue removed, and thus protects the points of least resistance from future microbic infection, caries, and necrosis.

Practical experience has demonstrated that in a large majority of cases the foregoing results are obtained. In some cases the middle-ear cavity becomes quite dry and free from the secretion of pus and mucus. In others, mucus and a small amount of pus remain as a more or less constant symptom. The mucus is oftentimes, no doubt, thrown into the middle-ear cavity from the Eustachian tube, especially if there is stricture of the tube near its lower extremity. Appropriate attention to this condition may cause a cessation of the mucous discharge. In those cases in which the general health is debilitated by some general constitutional disease, as scrofula, the middle ear will continue to discharge more or less thin, slimy pus, which shows a tendency to undergo ammoniacal decomposition. The adminstration of the iodides, iron, and arsenic will afford more or less relief.

In a general way it may be said that after the operation described there is but slight probability of a return of acute mastoid symptoms or of the extension of the necrotic process, as the non-vascular fibrous tissue which forms in the place of the bone removed affords protection to the points previously weakened by degenerative processes. In other words, the liability to the development of meningitis, brain-abscess, and thrombosis of the lateral sinus is reduced to the minimum.

Ear Disease as Affecting Longevity.

The casualities attending ear disease may be studied under two captions, namely: (*a*) accidental death on account of deafness, dizziness, and tinnitus; and (*b*) death due to the pathologic processes attending the diseases.

Accidental Death

may result from (*a*) **deafness,** as the one thus affected cannot hear the approach of railway trains, street cars, and other moving objects. Many deaths occur annually on this account; hence marked deafness is considered a bar to the issuance of life insurance. One affected by (*b*) **tinnitus** may confound the subjective noises in his head with those of an approach-ing railway train or other moving objects, and thus subject himself to considerable risk of death from traumatism. (*e*) **Dizziness,** or **aural vertigo,** may lead to accidental death by confusing the mental faculties, or by causing prostration of the patient at a time or place which places his life in jeopardy.

As most ear diseases are attended by one or more of the foregoing phenomena, their importance in estimating life expectancy becomes very great. **Mild deafness and tinnitus** should not, however, be regarded as sufficient grounds for the rejection of an applicant, unless the history of the case shows rapid progress of the disease. **Stricture or other stenosis of the Eustachian tube, dry and adhesive catarrhs of the middle ear, chronic and acute suppurative inflammations, perforation of the drum-head,** and **cholesteatoma,** etc., are attended by all these phenomena, and should receive most careful consideration in estimating the life expectancy. **Inflammation of the auricle and external auditory meatus, eczema,** and **inspissated cerumen** cause temporary slight deafness and tinnitus, and have little significance in this connection except as the eczema or inflammation may point to more serious involvement of the tympanic and mastoid cavities. **Ankylosis** and **adhesions of the foot-plate of the stapes** cause deafness and pronounced tinnitus. **Inflammation** and **degenerative changes of the membranous labyrinth** also cause marked aural disturbances, and may, therefore, lead to death from accidental causes. It may be said, in a general way, that diseases of the labyrinth are attended by more pronounced disturbance of the auditory functions than disease of the middle ear, and their presence should be regarded as more grave on that account.

One year of freedom from the disease after treatment or operation should entitle the applicant to another chance to pass the examination for life insurance.

Diseases of the Ear which May Affect Longevity.

The following classification is arbitrary, and should be regarded as suggestive rather than final. The first class will include those diseases which **should not bar** the applicant from receiving the benefits of life insurance; the second class includes those diseases which render the applicant **a doubtful risk**; and the third class, those diseases which render him **an unsafe risk.**

Diseases which Should Not Bar the Applicant from Life Insurance.

1. Malformations of the auricle. 2. Benign tumors of the auricle. 3. Eczema. 4. Furuncles. 5. Cerumen. 6. Otomycosis of meatus and membrana tympani. 7. Low perforations of the drum-head. 8. Calcareous deposits in the drum-head. 9. Mild non-progressive chronic otitis media. 10. Mild deafness from tubal catarrh. 11. Anæmia of the labyrinth, as a local expression of a mild general anæmia. 12. Hysterical deafness. 13. Diplacusis.

Diseases which Render the Applicant a Doubtful Risk.

1. Rapidly increasing deafness from any cause. 2. A shining, glistening external meatus is a suspicious sign of sclerosis of the middle ear, or even of the labyrinth; hence careful examination should be made before passing the applicant. 3. Pronounced stenosis of the Eustachian tube. 4. Over-permeability of the tube, as it is significant of sclerosis. 5. Simple hyperæmia of the labyrinth if it is due to pulmonary, renal, or cardiac lesion, is grave, and should relegate the applicant to the third class. 6. Perforation of the membrana flaccida (as it is a sign of attic, and possibly of antral and mastoid disease).

Diseases which Render the Applicant an Unsafe Risk.

1. Auricular erysipelas. 2. Gangrene and phlegmon of the meatus and auricle. 3. Perforation of the membrana flaccida attended by a foul-smelling, dirty discharge. 4. Abscess beneath the mastoid periosteum. 5. Inflammation and bulging of the postsuperior wall of the meatus. 6. Chronic perforation in the postsuperior quadrant of the drum-head. 7. Cholesteatoma. 8. Recurrent pains over the mastoid with chronic pus discharge from the meatus. 9. Aural polypi and

granulations. 10. High perforation of the drum-head. 11. Pinhole perforations at the margin of the drum-head (significant of tubercular disease). 12. Necrosis of the ossicles. 13. Chronic suppuration with destruction of the entire drum-head, the remnants of the ossicles being buried in a mass of granulation-tissue in the floor of the attic. 14. Malignant disease. 15. Tubercular disease of the tympanum. 16. Stenosis of the external meatus following chronic discharge or the mastoid operation. 17. Ankylosis of the foot-plate of the stapes. 18. Anæmia of the labyrinth due to tuberculosis. 19. Nausea and vomiting attended by a rapid pulse may indicate renal, cardiac, or pulmonary disease, and should be looked upon as grave signs. 20. Ménière's symptoms. 21. Ménière's disease (much more grave). 22. All inflammations of the labyrinth either from extension from the middle ear or from syphilitic, tubercular, or exanthematous infections. 23. Malignant neoplasms.

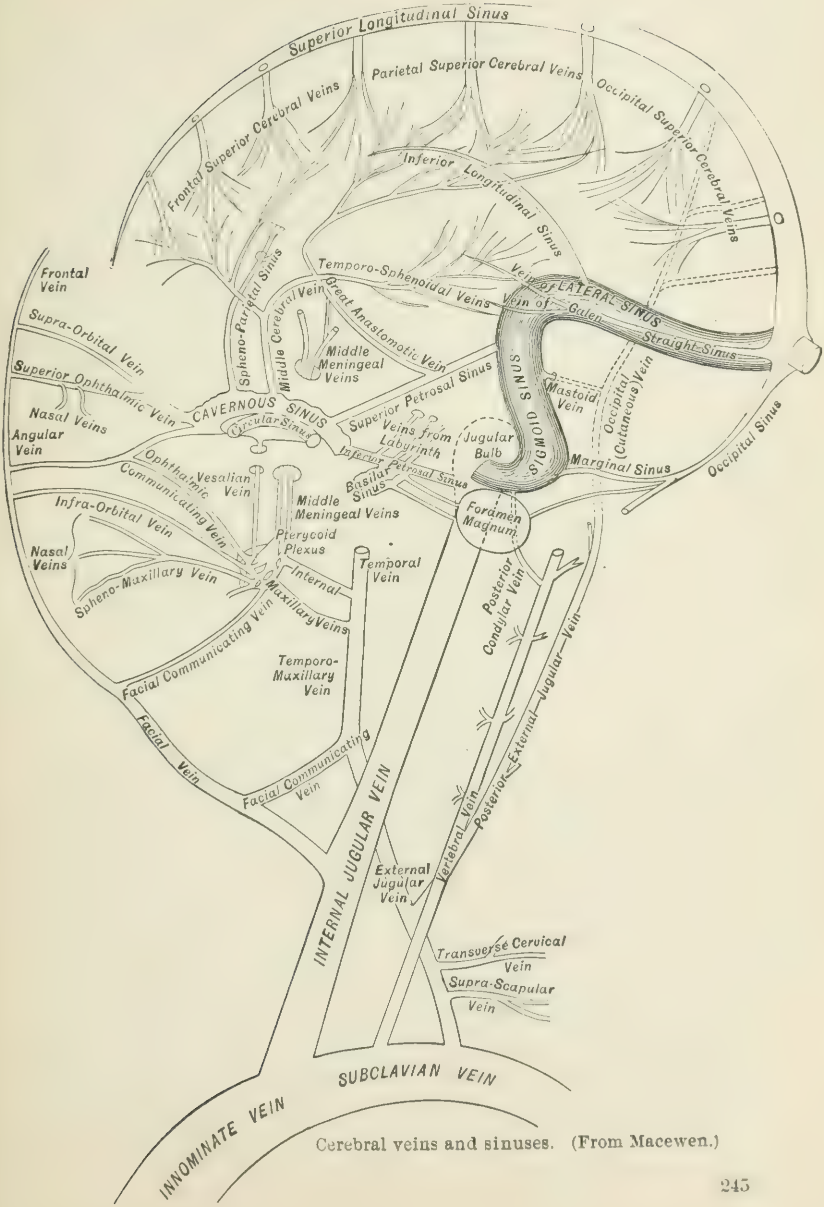
Thrombosis of the Sigmoid Sinus.

Etiology and complications: Thrombosis of the sigmoid sinus may occur in the course of chronic suppurative inflammation of the mastoid cells, either from close proximity or venous communication (Fig. 66). It may be aseptic or septic. If the thrombus disintegrates and spreads throughout the blood, septicæmia or pyæmia results. The particles of floating thrombus may lodge in the lungs, liver, kidneys, or other parts of the body, and give rise to septic foci of inflammation. In the course of thrombosis of the sigmoid sinus it has been often observed that there is a rusty sputum, and in some cases a gangrenous odor. Purulent basic leptomeningitis may occur on the median side of the sinus, and there may be a cerebellar inflammation with abscess.

If the thrombus is aseptic, it may be absorbed and leave the sinus thickened, contracted, or obliterated.

Thrombosis of the sigmoid sinus—symptoms: Severe rigors and convulsions at intervals of two or three days, followed by profuse perspiration, are the most characteristic symptoms. A high but *variable* temperature is present during the height of the process. A rapid and increasingly weak pulse marks

FIG. 66.



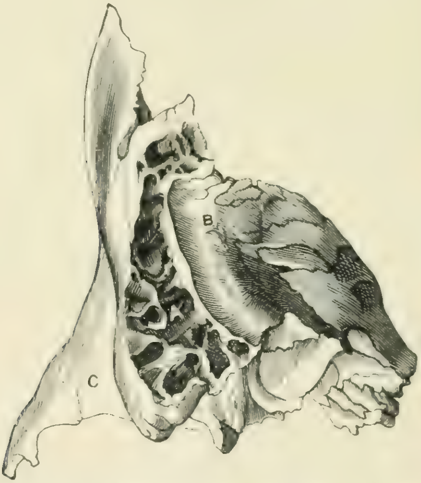
Cerebral veins and sinuses. (From Macewen.)

the progress of the disease. The pulmonary signs already referred to, and vomiting and diarrhœa, are also symptoms of importance. Giddiness is sometimes present, and the intellect remains clear in cases uncomplicated with meningitis. Pain on percussion and pressure is present on the posterior part of the mastoid. Œdema due to obstruction in the emissary veins of the skull is sometimes present.

Septic thrombi occurring in the other venous sinuses of the skull give rise to symptoms peculiar to their anatomical relations.

Prognosis: It usually runs a course of two or three weeks, and unless operative interference is practised terminates fatally.

FIG. 67.



Mastoid portion of the left temporal bone laid open and viewed from behind: *A*, mastoid cells extending from the mastoid process below, upward, and inward, over the lateral sinus *B*; *C*, the zygoma. (Burnett.)

Metastatic abscesses in the lungs, liver, spleen, and kidneys render the prognosis almost certainly fatal. It therefore becomes important that an early diagnosis and operation be made in order to remove the clot before it disintegrates and infects other organs of the body.

Thrombosis of the sigmoid sinus—treatment: The mastoid operation (described elsewhere) should be done, the sinus exposed along its mastoid portion (Fig. 67), and the fibrous wall of the vessel freely slit. The clot should be thoroughly removed, the wound packed with iodoform gauze, and the outer pushed against the inner wall and allowed to become adherent, and thus close the sinus. Another mode of operating is to ligate the jugular vein in the neck and then remove the clot. These operations are attended by the cure of many cases which would otherwise prove fatal.

The **lateral, superior petrosal,** and **cavernous sinuses** are more rarely involved. Impaired vision, exophthalmos, and œdema around the eyeball are signs of cavernous sinus thrombosis.

Meningitis of Otic Origin.

A much **larger proportion** of the reported cases of meningitis have their origin in chronic suppurative otitis media than is generally supposed. The author thinks it quite within the facts to state that one-half of the cases (excluding the epidemic and tubercular varieties) are due to suppurative ear disease.

There is comparatively **little danger** of meningitis (except in children where the bones are not fully ossified) until the suppurative process has continued intermittently for two or more years.

It is in the **old chronic cases,** in which the discharge has been scanty, that cranial involvement is most apt to occur. The reasons for this are given under *pathology* (p. 222). The discharge from the ear may have been so slight as to have escaped the notice of the patient or family physician, and yet a careful inspection would show pus which has become dried and exfoliates with epithelial scales of the external meatus.

The author wishes to **emphasize the fact** that a large number of the cases of meningitis are due to the so-called " mild suppurative ear discharges." They *seem* mild, but are in reality the *most dangerous* type of " discharging ears."

Leptomeningitis of Otic Origin.

Definition: This is a serous or purulent inflammation beginning in the arachnoid and extending to the contiguous membranes and superficial portion of the brain. It is an exceedingly fatal disease, and may run a rapid course, although in some cases it extends over several weeks.

The **symptoms** are headache, restlessness, insomnia, photophobia, retracted abdomen, contracted pupils, hypersensitiveness of the skin, full, bounding, rapid pulse, high temperature, and optic neuritis. As the disease progresses the intellect becomes dulled, the pulse slow, and the respiration rapid. Paralysis finally supervenes, and the urine and fæces are discharged involuntarily. The later signs point to a speedy termination.

Leptomeningitis—surgical treatment: In addition to the usual remedies, the mastoid may be opened as described and the middle cranial fossa, the lateral sinus, and neighboring parts explored and freed from necrotic and other morbid material. Irrigation and drainage sometimes avert a fatal issue. The operation seems justifiable at times, as without it a cure can hardly be hoped for.

Extradural Abscess.

Etiology: When in the course of chronic suppurative inflammation of the attic, antrum or mastoid cells, the bony walls become necrosed and perforated, pus-pockets may form between the dura and the skull.

Treatment: They should be thoroughly exposed and irrigated with bichloride solution (1:3000). The preliminary step in the operation is the radical mastoid operation. When this operation is done, intracranial abscesses are found that would have otherwise escaped observation until more serious involvement occurred.

Cerebral and Cerebellar Abscess.

Etiology: This may be a later manifestation of an extradural abscess which has eroded through the dura and penetrated the brain, or it may be due to a thrombus carried by the small cerebral arteries. Infection may also gain entrance through the perivascular sheaths. A few cases have been reported as occurring in children where infection has taken place through the petrosquamosal suture. Tubercular disintegration of the petrous portion of the temporal bone leads to tubercular abscess of the temporosphenoidal lobe, and is usually associated with perforation of the roof of the attic or antrum. This is important to remember, as in operating it is desirable to get at the seat of the trouble, remove all morbid material, and establish free drainage through the middle ear.

In mastoid operations extreme care should always be taken to examine the roof of the attic and antrum, as well as the walls of the lateral sinus, otherwise the wound will sometimes be closed and the existing brain or extradural abscess overlooked until serious and perhaps fatal brain disease has developed.

Cerebral and cerebellar abscess—symptoms: Oftentimes the first sign is the cessation or partial suppression of the ear discharge. There may be nausea, vomiting, pain, and mental irritability. Chilly sensations may be complained of. The normal temperament is often reversed—that is, a vivacious, socially inclined person becomes morose and seclusive, and *vice versâ*. After a time cerebration becomes dull, the pulse quite slow, and the temperature subnormal or but slightly elevated. Occasionally there may be high temperature, rapid pulse, rigors, and convulsions, as in thrombosis of the sigmoid sinus.

Aphasia in this connection is an almost certain sign of abscess of the temporosphenoidal lobe. In right-handed people it is a sign of abscess on the left sde, while in left-handed persons it is a sign of abscess on the right side.

There may be optic neuritis in cerebellar abscess, and, rarely, in abscess of the cerebrum. In cerebral abscess the percussion-note over the mastoid is high. This is due to the sclerosis which has occurred in the mastoid as a result of the chronic suppurative inflammation. This also accounts for the perforation being upward through the roof of the attic and antrum rather than backward through the mastoid process.

The reflexes of the limbs on the opposite side from the abscess are usually increased.

The characteristic symptoms of abscess in the *cerebellum* are slow pulse, low temperature, headache, nausea, vomiting, staggering gait, vertigo, and facial paralysis. Bacon reports a case of caries of the semicircular canals which presented the above symptoms; hence the diagnosis is not always easy. The establishment of free drainage is sometimes followed by relief of the symptoms of intracranial abscess. This does not necessarily prove that brain abscess did not exist, as it may drain through the perforation in the roof of the attic or antrum, after the removal of the granulations which obstructed it.

Sometimes the Cheyne-Stokes respiration, apathy, intolerance of light, yawning, and rigidity of the muscles of the neck and jaws are present.

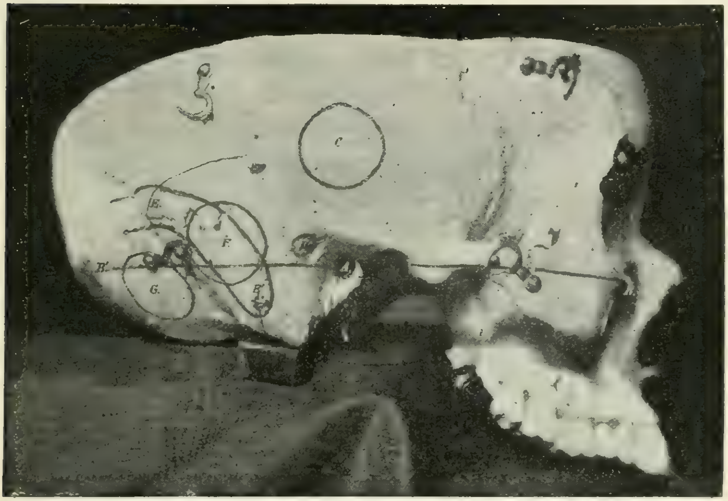
Prognosis: Without operative treatment nearly all cases end fatally. Under operative treatment about 30 to 50 per cent. recover.

Cerebral and cerebellar abscess—treatment: It is usually best to do the mastoid operation, converting the middle-ear cavity, attic, antrum, and mastoid cells (if need be) into one large cavity which can be freely inspected and explored for caries and perforation. If the temporosphenoidal lobe is the seat of the abscess, a perforation will usually be found through the roof of the attic or antrum. If possible, the abscess should be searched for through the perforation and its contents drawn off with a syringe, the needle of which is at least 1 mm. in diameter. If it cannot be reached through the point of perforation, the skull should be trephined at *C* in Fig. 68.

Through this opening the needle should be introduced and part of the contents of the cavity withdrawn, which should then be laid open with a scalpel, care being taken to avoid cutting the arteries, and irrigated with a warm 2 per cent. solution of boric acid. All sloughs should be carefully removed with a spoon-curette before closing the wound. A strip of sterilized boric-acid gauze should be introduced loosely into the abscess-cavity for drainage. This should be changed daily until the amount of discharge materially decreases. A 1 : 5000 bichloride solution may be used with advantage when the cavity is the seat of chronic streptococcal infection.

When the abscess is in the *cerebellum*, the mastoid operation should also be performed, and the lateral sinus examined and freed from clots if they are present. The cerebellum should then be exposed by removing the bone around the sinus or by removing a button of bone at the point *G*, Fig. 68. The pus should be drawn off with a hypodermic syringe or the cavity freely laid open with a scalpel, irrigated with warm boric-acid solution, and a loose gauze dressing introduced.

FIG. 68.



A, external auditory meatus; *B B'*, Reid's base-line running from the lower margin of the orbit through the centre of the external auditory meatus; *C*, trephine-opening, three-quarters of an inch in diameter, to expose temporosphenoidal abscess, the centre-pin of the trephine being placed one and one-quarter inches above the centre of the external bony meatus; *D*, point at which the mastoid antrum should be opened; *E E'*, position of sigmoid sinus; *F*, trephine-opening, three-quarters of an inch in diameter, for exposing sigmoid sinus, the centre-pin of the trephine being placed at a point one inch behind and one-quarter of an inch above the centre of the external bony meatus; *G*, trephine-opening for cerebellar abscess, the centre-pin being placed two inches behind and one-quarter of an inch below the centre of the external bony meatus.

Cholesteatoma.

Definition: This is a disease usually occurring in the course of chronic suppuration of the middle ear, although rare cases have been reported in which there was no suppuration, and in which there was no evidence that such a condition had ever existed. It is *characterized* by an accumulation of flat epithelial scales arranged in concentric layers and admixed with cholesterin and pus. It is sometimes classified as a tumor. Its origin is not well understood.

The **symptoms** are those of chronic otorrhœa with the presence of whitish yellow masses of scales in the middle ear, attic, and antrum. Sometimes the bony walls of the antrum and mastoid cells are absorbed from the pressure exerted by the mass.

Treatment: The cholesteatomatous masses should be removed either through the external auditory meatus or through the antrum after exposing it by a mastoid operation. If the mass is small and limited to the middle ear, it can be removed through the auditory meatus with curettes and irrigation.

External Mastoiditis; Superficial Periostitis of the Mastoid.

This condition is rarely primary, but is usually secondary to suppurative otitis media.

It is **characterized** by marked fulness and œdema on the affected side. The ear stands out quite prominently from the side of the head. There is nearly always furunculosis of the external auditory meatus. This will aid in the diagnosis between mastoiditis and this affection.

Treatment: A free incision should be made through the skin and periosteum, otherwise the bone beneath may become carious and the process extend to the mastoid cells. Recovery usually takes place rapidly after the incision is made.

Otalgia.

Etiology: There are no inflammatory lesions present to account for the pain. It may be due to a general neuralgic affection of the fifth nerve and may appear off and on for years. It may be reflex in origin from caries of the teeth and ulceration of the pharynx or larynx. It may be due to pressure upon the nerve. Catarrhal thickening of the mucosa of the middle ear may also excite it.

Otalgia—treatment: Examine the teeth, larynx, and pharynx for caries and ulcerations, and if a lesion is found, remove it. Examine the middle ear for catarrhal thickening of the mucosa, and if present attempt to reduce it by appropriate treatment. If syphilis is suspected, give the iodides or mercury according to the stage of development. Anæmia should be overcome by appropriate attention to the lower bowel and the administration of iron and arsenic. Local remedies sometimes afford great relief, such as mild stimulating liniments applied over the mastoid, or warm olive oil dropped into the meatus. Chloroform vapor blown into the ear with a clay pipe or other instrument sometimes causes instant cessation of pain. Constitutional dyscrasiæ should be corrected or ameliorated as much as possible. Nutritional activity in the middle ear should be excited by the use of pneumatic massage in the external auditory meatus. The treatments are best administered with a pump propelled by an electro-motor engine as described under Dry Catarrh. The hand soon tires in using the Delstanche masseur.

Contractions of Muscles.

Spastic contractions of the muscles of the auricle: They usually occur in connection with facial spasm (tic convulsif).

Spastic contractions of the tensor tympani muscle: They are characterized by movements of the membrana tympani and a loud, crackling noise, or a dull throbbing in the ear. The noise is often perceptible to the observer.

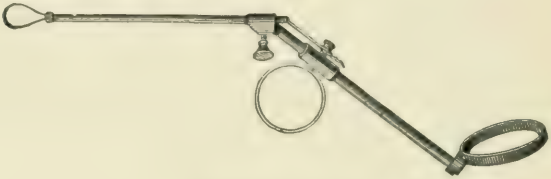
Clonic spasm of the muscles of the Eustachian tubes: A sound as of a distant, irregularly ticking watch may be heard, which is synchronous with the contractions of the velum palati.

Aural Polypus.

Varieties: They may be true myxomata, fibromata, angiomata, or œdematous granulation-tissue. They are usually the latter or of the fibromatous type, and are often significant of caries and necrosis in the attic or antrum. They obstruct the drainage of the mastoid cells and antrum into the middle ear, and by pressure cause absorption of contiguous bony tissue. They may also prevent the extension of infection through necrotic areas in the attic and antrum.

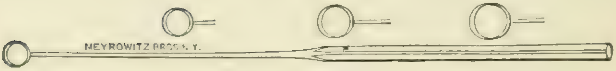
Treatment: They should be removed either with a small wire snare, curettes (Figs. 69 and 70), caustics, the galvanocautery, or by the instillation of 95 per cent. alcohol.

FIG. 69.



Blake's polypus snare.

FIG. 70.



Sharp curettes.

When the polypi are pedunculated and protrude into the external auditory meatus, they are best removed with Blake's snare.

If they are large, the galvanocautery applied carefully causes them to slough and shrink. Chromic acid crystals fused on the end of a probe and applied to the growths is a useful method of treatment.

When the granulations are small and succulent, alcohol instilled into the middle ear for ten or fifteen minutes every second day causes them to shrivel, and checks their growth. This is a useful adjunct to the other methods of treatment.

The meatus should be packed with sterilized gauze after any of the above methods of treatment.

It should be borne in mind that polypi or granulations usually grow from the upper part of the tympanum, probably the roof of the attic, and that the bone from which it grows is probably softened or perforated by caries. Careless or rough curettement might, therefore, open the cranial cavity and expose it to the danger of infection. The granulations are barriers to infection, within certain limits, and they should not be thoughtlessly removed. It is often a better plan to open the antrum as described under Mastoid Operation, and thus eradicate the entire diseased tissue, and establish instead firm non-vascular fibrous tissue, which will effectually protect the weakened areas.

Incision of the Drum-head (Paracentesis).[1]

General indications for the incision of the drum-head: (*a*) To afford an exit for serous, catarrhal, purulent, and other fluids from the middle-ear cavity.

(*b*) To relieve pain due to tension.

(*c*) To remove pressure from the labyrinth.

(*d*) To open the middle-ear cavity for certain operations.

(*e*) To allow sound-waves to reach the oval and round windows for the purpose of improving hearing.

Incision in the secretive form of catarrh: It is indicated as follows:

(*a*) If after several days' treatment the exudate does not decrease.

(*b*) When no exudate is seen and Politzerization is followed by relief for a short time.

(*c*) When the exudate is copious, paracentesis may be done without preliminary treatment, thereby hastening the cure.

Incision in acute myringitis: (*a*) In *abscess-formation* between the layers of the membrane, incision should be made to prevent spontaneous evacuation of the pus into the middle-ear cavity. Care should be exercised to avoid incising the entire thickness of the drum-head.

(*b*) When *pearly-gray blisters* are seen upon the drum-head, they should be pricked if they have not already spontaneously discharged their contents.

(*c*) *Inflammation* of the *deeper layers*, with bulging reddish or bluish swellings, should be immediately lanced to relieve pain and tension. Care should again be exercised not to penetrate the entire thickness of the drum-head.

Preliminary incision of the drum-head in tenotomy: The incision is made as a preliminary step in tenotomy of the tensor tympani and the stapedius muscles.

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[1] Published by the author in the *Plexus*, the official organ of the College of Medicine of the University of Illinois.

Incision for thickened drum-head: The incision is indicated in this condition when it is desirable to relieve the tension upon the labyrinth, and to allow sound-waves to reach the oval and round windows. Effort should be made to establish a permanent opening in the drum-head.

Incision in acute inflammation of the middle ear: (*a*) To relieve the severe pain, attended with or without fever, which resists other treatment.

(*b*) Circumscribed red spots or protuberances on the drum-head should be incised.

(*c*) When the most bulging portion of the drum-head is of a greenish-yellow color, it should be freely lanced.

(*d*) In excessive livid swelling of the cutis (outer layer), superficial scarification often relieves pain.

Incision in acute suppuration of the middle ear: (*a*) A bulging drum-head, with greenish color, severe pain, fever, and brain symptoms, calls for immediate incision.

(*b*) When there are symptoms of mastoid involvement, it should be incised to establish free drainage.

(*c*) Severe pain, unaccompanied by bulging, may be relieved by incision.

(*d*) Symptoms of pus-retention after closure of the primary opening are relieved by free incision.

Incision in adhesive processes in the middle ear: (*a*) To allow sound-waves to reach the labyrinth in cases in which there are ankylosis and adhesions which interfere with the vibration of the membrana tympani and ossicles.

(*b*) To restore equilibrium of air-pressure in the middle ear.

(*c*) In extensive calcareous deposits, and in abnormal thickening and retraction of the drum-head, paracentesis is sometimes indicated.

(*d*) In fixation of the malleus and incus by immediate ligamentous union with the walls of the tympanic cavity, tympanotomy should be done to expose the adhesions to operative interference.

(*e*) In stricture of the Eustachian tube, which cannot be relieved by electrolysis or dilatation with the Eustachian bougies, an effort should be made to establish a permanent opening in the drum-head. In this way the persistent retraction from diminished air-pressure will be overcome.

(*f*) In excessively loud subjective noises, if they cannot be relieved by other methods.

Incision in atrophy and relaxation of the drum-head: Superficial scarification may aid in restoring the drum-head to its normal tension.

Incision for diagnostic purposes: (*a*) In chronic suppurative otitis media, when the perforation is small and high, an incision may be made to enlarge the existing perforation, thereby facilitating a more thorough drainage and a more thorough exploration with a probe for roughened bone.

(*b*) Before performing ossiculectomy in sclerosis of the middle ear it is desirable to ascertain whether the operation will probably be followed by good results. For this purpose a preliminary V-shaped incision should be made and the hearing tested for improvement. If the incision is followed by improvement, there is some probability that the more radical operation will also be followed by improvement.

Preliminary examination of the function of hearing should be made before performing paracentesis for adhesive processes and dry catarrh; and ossiculectomy. Bone-conduction for watch and tuning-fork should be good, otherwise but slight improvement should be expected to follow the operation.

Technique of Paracentesis.

Electrocautery paracentesis in adhesive, non-inflammatory cases:

(*a*) Cocainize with a 20 per cent. solution for twenty or thirty minutes.

(*b*) The electrode should be a straight, simple pointed one, with the shank bent at such an angle as to offer no obstruction to the view of the operator.

(*c*) Instantaneous red heat should be obtained, in order to prevent the operation being painful to the adjacent tissues.

(*d*) Contact with the drum-head should be made before closing the circuit.

(*e*) Time of heat contact: from one-half to two seconds.

(*f*) The pressure should be very slight, otherwise the inner wall of the tympanum may be injured.

Incision with the lancet: (*a*) *Instruments:* A double-edged or spear-shaped lancet was formerly used, as only simple paracentesis was desired. Preference should be given, however, to Hartmann's curved lancet, as a free incision affords greater relief.

(*b*) *Anæsthesia:* When the drum-head is bulging from fluids in the tympanum, the incision may be made without the use of a local anæsthetic. In other conditions an instillation of a 20 per cent. solution of cocaine should be made from twenty to thirty minutes prior to the operation.

(*c*) *Location:* The most suitable location for the incision is usually in the postinferior quadrant, as it is more accessible and farther removed from the inner tympanic wall. If *fluids are present*, the bulging portion of the membrane should be incised.

(*d*) The *length of the incision* should be from two to four millimetres.

Points of interest after paracentesis: (*a*) Immediately after the incision is made a pulsation, or movement synchronous with the act of swallowing and articulation, occurs at the point of the incision. Pus or mucus rarely discharges into the meatus until after the lapse of several hours.

(*b*) Politzerization will force the secretion into the external meatus. In some cases the mucus is so tenacious that it cannot be removed by this method. Suction by means of the Delstanche masseur will usually succeed in removing it. Air may be compressed in the external meatus, thereby forcing the mucus out through the Eustachian tube. Strings or plugs of very thick mucus may be removed with the forceps. Instillations of warm soda solution or 15-volume hydrozone act as solvents and facilitate its removal.

(*c*) Closure of the wound usually occurs within one or two days in non-suppurative cases.

(*d*) *Dressing:* The external meatus should be packed with gauze or sterilized cotton, after thoroughly asepticizing the parts.

THE INTERNAL EAR.

Injuries to the Labyrinth.

Etiology: Injuries to the internal ear may occur from *direct violence*, as gunshot-wounds; and sharp instruments, etc., introduced accidentally through the external auditory meatus. The instrument may penetrate the oval or round windows and allow the intralabyrinthine fluid to escape.

Injuries from *indirect violence* may result from a blow to the skull, by which the petrous portion of the temporal bone is fractured; or from a blow upon the ear, in which the air is condensed in the meatus and middle ear and driven upon the oval and round windows. The terminal expansion of the auditory nerve is thus injured. A loud cannon-report may also cause condensation in the middle ear, and thus injure the terminal auditory nerve-endings.

The **symptoms** of concussion of the labyrinth vary with the degree of injury, from slight stupefaction and singing noises of a few hours' or days' duration, to immediate and marked deafness with loud subjective noises, confusion of ideas, and giddiness. After a time the subjective noises improve or change to a shrill metallic noise, especially in the presence of objective noises. The hearing often remains unimproved. Bone-conduction is much diminished or altogether wanting, according to the impairment of hearing. The vibrating tuning-fork on the vertex is heard best on the uninjured side.

The **prognosis** in most cases is unfavorable so far as a return of hearing is concerned. Sometimes there is improvement after many weeks or months; while in other cases there may be progressive increase in the deafness and subjective noises.

Hyperacuteness of Hearing.

Synonyms: Hyperæsthesia Acoustica; Oxyecoia.

Definition: This is a rare condition in which there is a *temporary increase* in the acuteness of hearing.

It may be **due to** intracranial irritation, local degeneration, the irritation occurring at the onset of infectious fevers, meningitis, hemicrania, and trigeminal neuralgia. Cases have been reported in which it occurred at the close of a mild catarrhal attack of the Eustachian tube and tympanic cavity. It is also quite common in severe forms of sclerosis and catarrh of the middle ear.

Within the last four years attention has been called to it as one of the signs of Amaurotic Family Idiocy.

Paracusis.

This is a condition of the auditory nerve or nerve-endings which is characterized by *perverted perception* of the pitch of musical sounds, sometimes amounting to one or two degrees. Slight changes in pitch may be due to altered tension of the sound-conducting apparatus; hence care should be exercised to differentiate the two conditions.

Paracusis loci is the inability to determine the direction of the source of sound. Sound upon the deaf side may be thought to come from the opposite, and may prove a source of danger.

Paracusis diplacusis: One ear having an altered tone perception, a sound is recognized by the two ears as a double tone usually quite discordant in character. Both ears may be unequally out of tune, and thus give rise to the phenomenon.

Paracusis Willisii: This is a condition occurring in the course of dry catarrh, or sclerosis of the middle ear, and is characterized by ability to hear better in the presence of objective noises, as in a moving railway train or on the street cars.

Subjective Noises in the Ear; Tinnitus.

The following quotation from Politzer (English translation by Dr. Dodd) gives in brief form the varying types of subjective noises experienced as the result of direct or reflex irritation to the auditory nerve:

"The subjective sensations of hearing are characterized by the patient in different ways—most frequently as whizzing, roaring, buzzing, seething, hissing, singing, ringing, humming, and whistling in the ear. The character of the noises is designated as either high or low by patients who are capable of judging. More rarely they are compared to the noise of a railroad train, the chirp of a cricket, the warble of birds; or (the most rare form of noises) articulated human voices, the barking of a dog, the crashing of plates of glass, the grinding of shears, the cracking of braces, the sound of trumpets, the tone of a low or high violin-string, chaotic musical tones, crashing and crackling, the shot of a pistol, the sensation of wind blowing outside the ear, the blow of a hammer, the noise of a mill, the croaking of frogs, etc. Frequently the *objective* noises (as the tick of a watch) are not heard as such if they are similar to the subjective noises of the patient."

Etiology and occurrence: The conditions of atmosphere and climate which render catarrhal diseases worse, increase the noises. They are usually worse at night and in quiet places. They may be intermittent or constant. They may occur in persons of a nervous, irritable temperament without accompanying deafness. This type usually continues throughout life.

Continuous noises are rarely cured, while the intermittent type is more favorable.

Subjective noises in the ear—treatment: The *cause* should be determined and removed or ameliorated if possible, such as existing external-, middle-, or internal-ear disease; coexisting nasal and nasopharyngeal diseases; anæmia or other constitutional disorders.

There are certain *local measures* which afford relief in a considerable number of cases. The most successful are inflation of the tympanic cavity by the Politzer method; inflation by catheterization; and pneumatic massage of the external auditory meatus with the Delstanche masseur or the electric pump. The injection of medicated oils into the tympanic cavity through the catheter is also sometimes attended by relief. Artificial perforation of the drum-head affords temporary relief in cases in which there is great retraction of the drumhead from closure of the Eustachian tube.

Hyperæmia, Anæmia, and Hemorrhage of the Labyrinth.

Hyperæmia is found in acute purulent inflammation of the tympanum; in the acute exanthematous fevers; in meningitis; and in diseases of the lungs and heart. The administration of the nitrite of amyl, salicylic acid, and quinine may also cause it.

The *symptoms* are giddiness, vertigo, staggering gait, or a tendency to turn into the gutter or against the side of the house, vomiting, tinnitus, and a feeling of fulness in the ears.

The *treatment* should be addressed to the cause.

Anæmia of the labyrinth: It may occur in general anæmia and chlorosis, after great loss of blood, and in tuberculosis. A rapid pulse with deafness, vertigo, nausea, vomiting, pronounced tinnitus, and faintness should excite suspicion of pulmonary tuberculosis. The *prognosis* in these cases should be guarded.

Hemorrhage into the labyrinth: It may *occur* from fracture of the base of the skull; severe concussions to the head or the external ear; necrosis of the petrous portion of the temporal bone; and from atheromatous changes in the walls of the bloodvessels of the labyrinth. The hyperæmia attending the exanthematous fevers may also give rise to slight hemorrhages.

Increased Labyrinthine Pressure.

Etiology: Catarrhal and adhesive processes in the middle ear may cause increased labyrinthine pressure by retraction of the drum-head; or by adhesive processes with contraction of the adhesive bands, whereby the foot-plate of the stapes is held forcibly against the oval window. Adhesive bands and calcareous deposits sometimes interfere with the expansile properties of the membrane of the round window, and may thus increase the intralabyrinthine pressure.

The **symptoms** are tinnitus, giddiness, deafness, unsteady or uncertain gait, and sometimes nausea and vomiting. This condition is often confounded with Ménière's disease, in which there is complete and sudden loss of hearing.

Paresis and Paralysis of the Auditory Nerve.

Origin: It may be of *angioneurotic, rheumatic*, or *hysterical* origin.

The **angioneurotic** type of auditory paralysis is rare, and is characterized by sudden pallor of the face, which is immediately followed by giddiness, nausea, tinnitus, vertigo, and deafness. After a few moments the pallor and ear symptoms disappear, leaving no deafness or trace of ear disease.

Rheumatic paralysis of the auditory nerve occurs occasionally in the course of rheumatism in other parts of the body. There is usually complete deafness, which gradually disappears.

Hysterical paralysis is characterized by an unstable and eccentric nervous system, and deafness which appears and disappears rapidly without visible signs of ear disease to account for it.

Paresis and paralysis of the auditory nerve—treatment: This will depend upon the duration of the deafness and the causes of the same. The more acute the case the more favorable is the result of the treatment. Rest in a quiet room and the use of hot foot-baths with mustard, vesicants over the mastoid, and saline purgatives often favor a rapid recovery. *Local treatment*, as inflation, etc., is contraindicated in most cases, especially when there are no signs of middle-ear disease.

Cerebral Disturbances of Hearing.

Etiology: Meningitis, cerebrospinal meningitis, cerebral hemorrhage, emboli, embolic softening, encephalitis, chronic sclerosis, acute and chronic hydrocephalus, gummata, tubercular nodules and tumors at the base of the brain may cause disturbances of hearing.

Inflammation of the Labyrinth.

Labyrinthitis; Otitis interna.

Pathology: The condition is usually secondary to inflammation in the middle ear, especially that following diphtheria, scarlet fever, and influenza. The lining of the bony labyrinth and the utricle and saccule are usually affected. In the *chronic* type there is an increase in the connective tissue with hyperostosis and deposit of calcareous salts. The utricle, saccule, and ampullæ may become thickened, and finally lead to atrophy of the membranous labyrinth and organ of Corti.

The **symptoms** of *acute* inflammation of the labyrinth are almost identical with those of meningitis as it occurs in children followed by recovery. The attack is more sudden and lasts for about a week.

Syphilis of the Labyrinth.

It usually occurs as a **tertiary manifestation.** There are infiltration of the membranous labyrinth, fixation of the foot-plate of the stapes, and condensation of the periosteum of the vestibule. It may be associated with a middle-ear catarrh, attended by thickening of the drum-head.

Symptoms: There are sudden deafness and tinnitus, with more or less vertigo and unsteady gait. Pain is usually absent.

The **prognosis** depends upon whether it is congenital or acquired, and recent or of long standing. The congenital type is less favorable, and the more acute cases are more favorable.

Treatment: Potassium iodide internally, and inunctions of mercury over the mastoid, are of value in recently acquired cases.

Meniere's Disease.

Symptoms: This is a disease of the semicircular canals, and may extend into the vestibule. It is characterized by sudden total deafness, followed by giddiness, great tinnitus, vertigo, staggering gait, and vomiting. There may be premonitory symptoms, as pallor, cold perspiration, etc. Bone-conduction on the affected side is very much diminished or entirely absent. Upon examination, the drum-head appears normal, unless it happens to have been affected in the course of a middle-ear disease. The body inclines to the affected side in walking. The giddiness and staggering gait disappear slowly. The tinnitus is usually quite persistent and distressing. The deafness rarely improves, and if at all, but slightly.

Meniere's disease—treatment: This seems to be of little value. Rest and quiet, with cold applications to the head and calomel catharsis, perhaps exert a slight favorable influence. Injections of 4 to 6 drops of a 2 per cent. solution of pilocarpin through a Eustachian catheter is one of the best remedies, although its use is not always unattended with danger. Those suffering with a weakened heart from any cause will tolerate its use badly. The remedy should be used once daily for a month. The patient should remain in bed and be placed upon a light diet. Quinine, potassium iodide, potassium bromide, and the dilute hydrobromic acid in 15-minim doses, relieve the giddiness. The author has used quinine in one case with gratifying results.

"**Meniere's symptoms**": In the course of exudative middle-ear catarrh, chronic dry catarrh with adhesions and contractions may force the stapes into the oval window and give rise to several of the symptoms of Ménière's disease.

The two conditions are distinct pathologic entities, and should not be confounded. "Ménière's symptoms" are not attended by complete sudden deafness and ear disease is present to account for the symptoms. Inflation of the tympanum is usually followed by marked relief or the disappearance of the deafness and other symptoms of the "Ménière group."

Varieties of Deafness; and Tumors.

Boiler-makers' deafness: This is characterized by deafness with impairment of bone-conduction. A change of occupation is sometimes followed by improvement.

Senile deafness: There is deafness with impaired bone-conduction, and lessened ability to hear the high notes of Galton's whistle or of the König rods. The deafness may also be due to thickening or atrophy of the drum-head, and to collapse of the cartilaginous meatus.

Tumors affecting the labyrinth: They are rarely primary, but are generally from the cranial cavity or middle ear.

Deafmutism.

Definition: Absence of the power of speech on account of congenital or acquired deafness is called deafmutism. If it is acquired, it must occur soon after birth, or before articulate speech is learned.

Etiology: Congenital deafness may be the result of arrested or imperfect development of the temporal bone. Acquired deafness in infants is usually the result of the exanthematous fevers, meningitis, cerebrospinal meningitis, injuries, and syphilis.

Examination: It is difficult to examine deafmutes on account of their age and lack of training. To test the hearing, the physician should stand where he cannot be seen by the child, and clap his hands loudly, ring a bell or gong, or blow a loud whistle. If the child hears, he will turn toward the noise or manifest by the expression of the face that he hears the sound. By placing the higher tuning-forks upon the vertex the author has in some cases noted a pleased smile come upon the face; while the lower (128 v.) ones caused a displeased look or expression of disgust. Patient and continued effort will usually enable the physician to determine the state of the acoustic nerve.

The author also uses the graphophone with the ear-tubes for testing mutes.

Prognosis: Unless the power of speech is well developed when deafness occurs, the child is likely to lose his speech. If deafness occurs after the seventh year, the child generally retains the faculty of speech. More particularly, the prognosis depends upon the age of the child, whether the deafness is congenital or acquired, and upon the specific cause or causes of the condition. Congenital cases are occasionally benefited by treatment, while the acquired type is rarely improved. The more recent the deafness the more favorable the prognosis.

If the child is intelligent, and is sympathetically, intelligently, and systematically managed, there is a possibility of teaching it many useful things, and of giving it a trained and cultured mind. The case of Hellen Keller is a remarkable instance illustrative of this point. The child was not only a deafmute, but was blind also. She acquired a college education and was able to compose essays of a quality of no mean order.

Deafmutism—treatment: This should have a twofold purpose, namely: (*a*) the removal of the cause and improvement of the deafness; and (*b*) the education of the child.

All catarrhal, adhesive, and other middle-ear disease should be most carefully treated, and the nasopharynx should also be freed from disease and the presence of growths and thickenings. Postnasal adenoids are often the cause of the deafness, and should be removed. The author has seen cases show marked improvement after their removal. Improvement in the faculties of speech and hearing does not always occur, but there may be an improvement in the nervous symptoms and the power to fix the attention.

It may be impossible to improve the hearing, while it is quite possible to improve the receptive powers of the mind. This phase of the subject is too often overlooked in the treatment of such cases. No pains should be spared to afford the much handicapped child every possible chance for mental culture and training. Schools and homes are now provided for the training and treatment of such cases, and there is a much more hopeful prognosis for them in consequence.

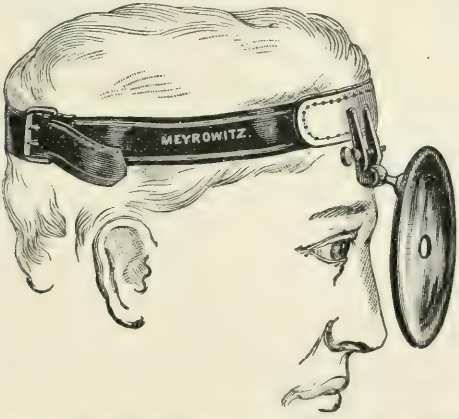
THE NOSE.

Illumination of the upper respiratory tract: The same sort of light may be used as described under examination of the ear. The mouth may be examined without artificial illumination or the aid of reflected light.

EXAMINATION OF THE NOSE.

The **instruments** (Figs. 71–78) needed for this purpose are a head-mirror, a nasal speculum, a probe, and postrhinoscopic mirrors ranging in size from No. 00 to No. 2.

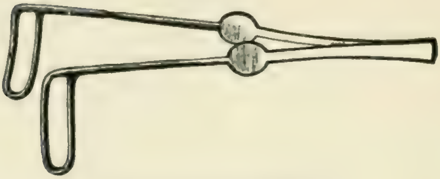
FIG. 71.



Head mirror and band, with its proper adjustment over the left eye.

To examine the **inferior turbinal** and **inferior meatus**, the patient should be directed to hold the head in the usual horizontal position, and not to throw it backward, as they instinctively do. The usual idea is that the interior of the nose runs upward and slightly backward. The floor of the nose runs directly backward when the head is in the normal upright

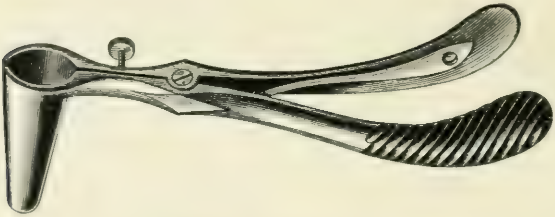
Fig. 72.



Bosworth's nasal speculum.

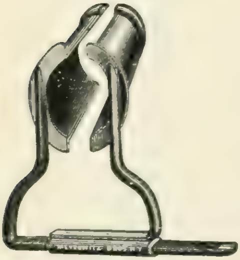
position. The tip of the nose should be lifted upward with the introduced speculum so as to clear the view of the interior. With the aid of the reflected light, the **inferior turbinal** should be examined for turgescence, hypertrophy, excrescences, collapse, atrophy, crusts, and synechiæ. If the body is swollen and pits freely upon pressure, it is turgescent and not hypertrophic. If it is enlarged and does not pit upon pressure, it is either hypertrophic or the seat of some neoplasm. In hypertrophy the contour of the turbinal is nodular, while in turgescence it is rounded or globular.

Fig. 73.



Coakley's nasal speculum.

Fig. 74.

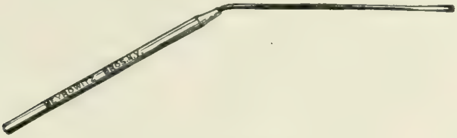


Myles' nasal speculum.

The **septum** should be examined for spurs, ridges, exostoses, enchondroses, deflections, ulcers, perforations, mucous hypertrophies, crusts, and other morbid conditions.

The **middle turbinal** and **middle meatus** may be examined by directing the patient to throw the head slightly backward, and proceeding as described for the examination of the inferior turbinal. Crusts, pus, clefts, polypi, necrotic areas, and enlargement from hyperplasia should be sought for.

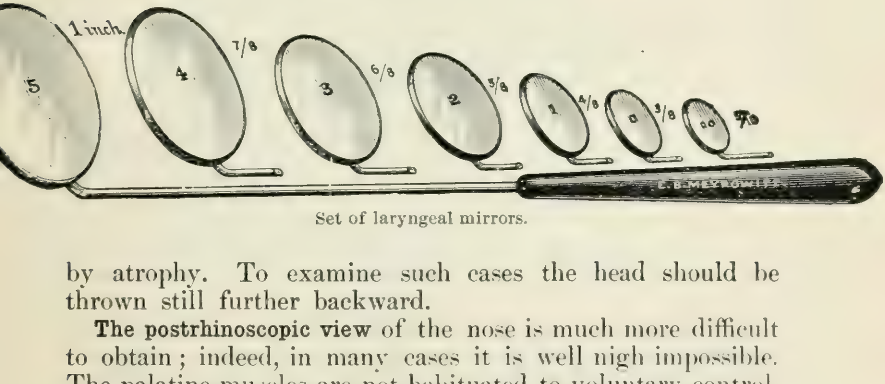
Fig. 75.



Nasal applicator.

The **superior turbinal** and **meatus** cannot be seen except in very rare cases in which the middle turbinated is destroyed by atrophy. To examine such cases the head should be thrown still further backward.

Fig. 76.

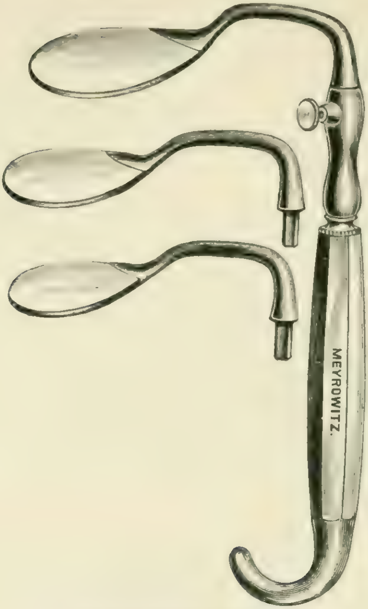


Set of laryngeal mirrors.

The postrhinoscopic view of the nose is much more difficult to obtain; indeed, in many cases it is well nigh impossible. The palatine muscles are not habituated to voluntary control, and the thought that something is being thrust into the throat excites spasmodic contraction of the muscles.

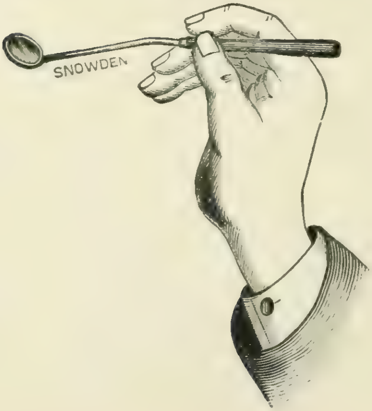
The tongue should be held down with a tongue-depressor (Fig. 77) and a small mirror introduced through the mouth. The mirror should be held as shown in Fig. 78. It should be introduced on one side of the uvula, as contact with the latter excites muscular spasm. By slightly raising, lowering, and rotating the handle of the mirror, all parts of the vault

Fig. 77.



Turck's tongue-depressor.

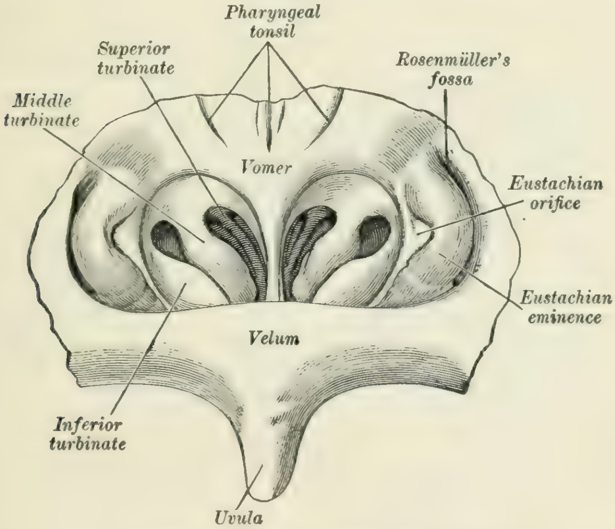
Fig. 78.



of the nasopharynx and posterior choanæ are brought into view. If the soft palate is drawn upward and backward against the wall of the pharynx, the patient should be encouraged to breathe softly through the nose. Repeated attempts may be required to accomplish a complete examination. The condition of the vault, mouths of the Eustachian tubes, posterior ends of the middle and inferior turbinals, and the posterior end of the septum should be noted (Fig. 79).

Examination of the base of the tongue: The tongue should be held as for the laryngoscopic examination and the laryngeal mirror held as for posterior rhinoscopy, except that the surface of the mirror should be directed downward. The root of the tongue is normally traversed by veins and comparatively loose folds of mucous membrane. In certain diseased conditions both tissues may be enlarged. There is lymphoid tissue here as in the vault of the pharynx, and this may be the seat of hypertrophy, although it most often occurs in adults contrary to the rule of lymphatic hypertrophy in children.

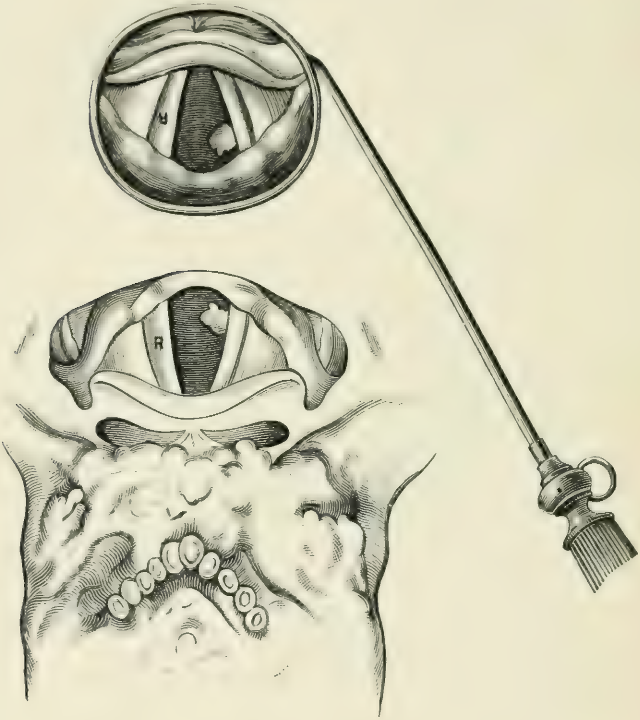
FIG. 79.



Posterior rhinoscopic image. (Schmidt.)

The larynx may be examined at the same time by slightly lowering the handle of the mirror.

Fig. 80.



Comparison of laryngeal image with specimen. (Schroetter.)

In all examinations with the laryngeal or postrhinoscopic mirrors the image is reversed (Fig. 80).

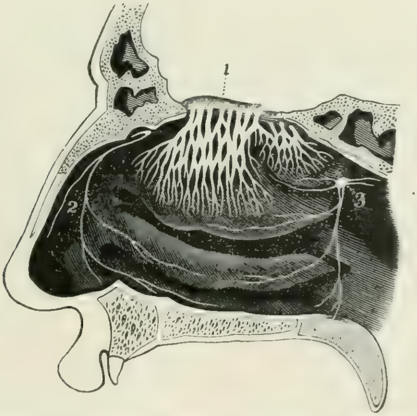
STRUCTURE OF THE NASAL MUCOUS MEMBRANE.

The nasal mucous membrane may be studied under two divisions, viz., the respiratory and the olfactory regions.

The membrane of the **respiratory region** is about 4 mm. thick over the inferior turbinals, and contains a network of veins giving it the general structure of cavernous tissue. The epithelium is stratified-ciliated-columnar in type. In the superficial layer are many goblet-cells. The second layer, the tunica propria, is composed of fibrous connective tissue, with here and there nests of leucocytes and lymphoid cells. The venous network is chiefly located in the tunica propria. The third layer is composed of submucous connective tissue in which numerous racemose glands are located.

The membrane of the **olfactory region** (Fig. 81) consists of two layers, the epithelial and the tunica propria. The epithelial layer contains the terminal bulbs and filaments of the olfactory nerve. The tunica propria contains the glands of Bowman, which were formerly thought to be serous, but which are probably mucous glands.

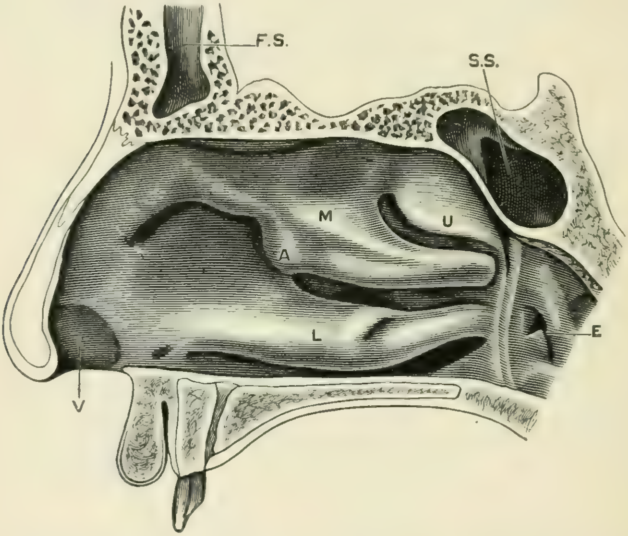
FIG. 81.



Distribution of nerves in the nasal passages (Dalton): 1, olfactory bulb, with its nerves; 2, nasal branch of the fifth pair; 3, sphenopalatine ganglion. (Seiler.)

"The swell bodies"; erectile tissue: Beneath the surface of the mucous membrane covering the lower and middle turbinated bones (Fig. 82) are large plexuses of bloodvessels. Zuckerkandl describes the mucous membrane covering the turbinated bodies as consisting of connective tissue, the outer layer covered with flat epithelium, the deep layer forming the periosteum of the turbinated bones. Between them are lymph-tissue, possibly lymph-glands, and an occasional tubular mucous gland. Within the lymphoid tissue there are abundant venous plexuses which he calls "swell bodies." About the plexuses are unstriped muscular fibres in abundance. The presence of the venous knots gives the tissue the character of erectile tissue, such as is found in the corpora cavernosa of the penis.

FIG. 82.



Anteroposterior section of the nose, showing the outer wall of the right nasal cavity: L, inferior turbinate; M, middle turbinate; A, anterior end of middle turbinate; U, superior turbinate; F.S., frontal sinus; S.S., sphenoidal sinus; E, Eustachian orifice; V, vestibule. (Zuckerkandl.)

The **arterial supply** is derived from the sphenopalatine artery. The *capillaries* are divided into three sets, viz.: (*a*) those going to the periosteum; (*b*) those going to the glands; and (*c*) those going to the surface of the membrane.

Those distributed to the surface form roots which empty into the veins, together with the superficial gland capillaries.

The deep gland capillaries and those of the periosteum empty into the veins, forming the so-called "swell bodies"; and the blood is then conveyed by venous channels in the periosteal surface of the membrane.

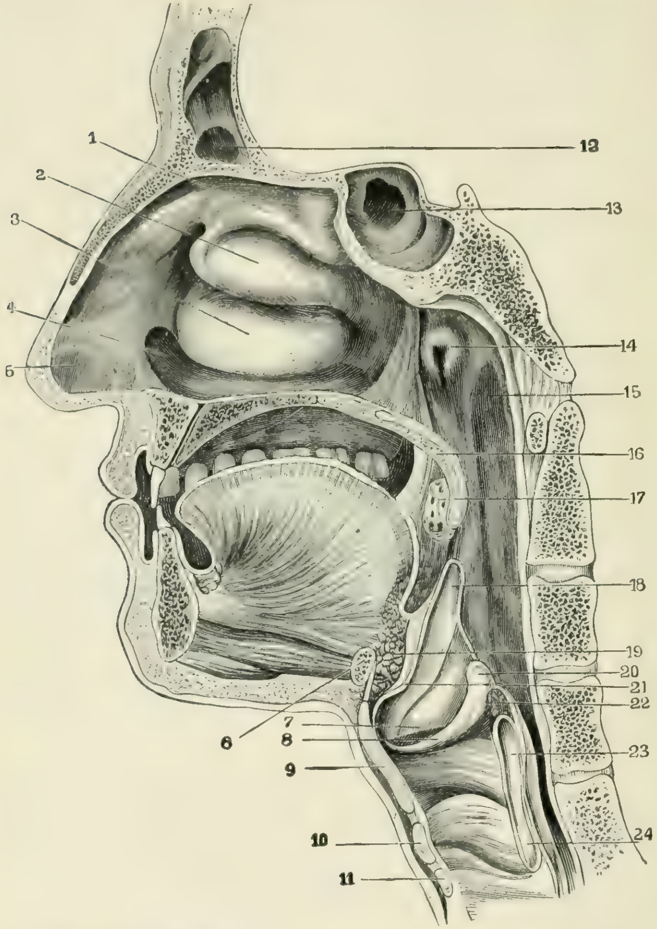
Distribution of the "swell bodies": (*a*) One is located along the free border of the inferior turbinated body; (*b*) a small one on the lower border of the middle turbinated body; (*c*) one on the posterior end of the superior turbinated body; (*d*) one on the posterior end of the middle turbinated body; and (*e*) one on the posterior end of the inferior turbinated body.

The function of the "swell bodies": Their chief function is to humidify and temper the air so that its presence in the lower air-tract will not excite irritation. The erectile tissue is controlled by the vaso-motor nervous system in such a way that the inhalation of cold, dry air causes an engorgement of the venous plexuses. In a normal nose the engorgement is not sufficient to cause nasal stenosis, but just enough to favor a more rapid transudation of serum, and an additional elimination of heat sufficient to raise the temperature of the inspired air to within one or two degrees of the body-heat. During the respiration of moist warm air, the "swell bodies" contract and throw out less heat and moisture. Ordinarily from fifteen to twenty ounces of serum are thrown out in twenty-four hours. This is absorbed by the warm and expanded air and carried to the lower air-tract, where it is needed for physiologic purposes. The trachea and bronchial tubes are supplied with but few glands, and are dependent upon the moisture thrown out in the nose (Fig. 83).

Certain *morbid conditions* interfere with the full performance of these functions, and lead to a train of symptoms more or less common in catarrhal conditions of the larynx, trachea, and bronchial tubes. The transfusion of gases in the air-vesicles of the lungs is also modified, and may lead to very serious nervous and nutritional disturbances from auto-intoxication.

Mucus secretion: The production of *mucus* by a normal membrane is carried on by the epithelial cells lining the mucous glands, and the superficial cells covering the membrane. The epithelial cells of the mucous glands secrete most of the mucus, but here and there on the surface of the

FIG. 83.



Vertical section of head, slightly diagrammatic: 1, superior turbinated body; 2, middle turbinated body; 3, lower turbinated body; 4, floor of nasal cavity; 5, vestibule; 6, section of hyoid bone; 7, ventricular band; 8, vocal cord; 9, section of thyroid cartilage; 10, 23, and 24, section of cricoid cartilage; 11, section of first tracheal ring; 12, frontal sinus; 13, sphenoidal cells; 14, pharyngeal opening of Eustachian tube; 15, Rosenmüller's fossa; 16, velum palati; 17, tonsil; 18, epiglottis; 19, adipose tissue behind tongue; 20, arytenoid cartilage; 21, tubercle of epiglottis; 22, section of arytenoid muscle. (Seiler.)

mucous membrane there are goblet epithelial cells which also pour out mucus. The mucus is made at the expense of the protoplasm of the cell-body, the process of production being somewhat as follows: the cells swell, the centres become transparent, and the protoplasm-granules are reduced to small strings or particles. The mucus thus formed is discharged and the cell-body left intact; or the entire cell may disintegrate and be thrown off with the mucus.

Mucus formation is a degenerative process whether it occurs under normal or pathologic conditions. Hence, in catarrhal inflammation of the nasal mucous membrane it is produced in nearly the same manner as that just described. Normally it appears as a thin viscid fluid, while that occurring in the course of catarrhal inflammation is oftentimes thick and tenacious. The difference is due to the diminished amount of serous transudation in inflammatory diseases of the membrane. The so-called normal mucous secretion is, in reality, a mixture of serum and mucus, and the amount of the two is greater than that found in diseased conditions, although the contrary seems to be true. In catarrhal inflammation the secretion is thicker and is not so readily absorbed by the inspired current of air. It must, therefore, be expectorated or blown upon a handkerchief. Patients often complain of an increased nasal discharge, and that there was a time when they did not need to use a handkerchief so frequently. The explanation lies in the fact that the solid elements of the mucus are increased while the serous element is much diminished: mucoid degeneration of the epithelial cells is an increased factor, while serous transudation is a diminished factor.

RHINITIS.

General Etiology of Rhinitis.

The nose as a filter: The interior of the nose is so constructed that the inspired current of air passes over a large moist surface of mucous membrane. There are about twenty-six square inches of surface thus exposed, and the dust and other foreign particles in the inspired air are deposited and discharged into the nasopharynx or through the anterior nares. The lower respiratory tract is thus protected from irritation (leading to inflammation) which the dust-laden air would otherwise produce. Germs are also filtered from the air and rendered non-pathogenic by the bactericidal properties of the nasal secretion.

The respiratory functions of the nose: From the above considerations it is seen that the respiratory functions of the nose are to humidify, temper, and filter the inspired air. The proper performance of these functions is of vital importance to the health of the individual. Further discussion of this topic is given under *postnasal adenoids.*

Anterior nasal stenosis: Partial closure or obstruction of the anterior nares causes the air back of the obstruction to be rarefied during inspiration, much as the air is rarefied in a syringe upon the sudden withdrawal of the piston-rod and valve. The blood in the mucous membrane of the nasal walls rushes in to fill the partial vacuum and *hyperæmia* is produced. The over-plus of blood leads to over-nutrition or hypertrophy of the parts. The character of the secretion is changed, and finally a slight or low-grade irritation results, which may lead to the formation of elastic fibrous tissue, which in extreme cases may amount to a true sclerosis (fibrous rhinitis).

Mouth-breathing leads to very marked nutritional and nervous phenomena. This topic is more fully discussed under *postnasal adenoids.*

The accessory sinuses of the nose: Prolonged inflammation of the nasal mucous membrane may be due to an inflammation of the mucosa lining the *frontal, ethmoidal,* or *sphenoidal cells.*

The discharge may be either mucus or mucopus. If the sinusitis is catarrhal, the secretion will be mucus; but it may be so changed in its chemical composition as to be irritating to the nasal mucous membrane. Catarrhal sinusitis is not usually recognized in practice, as the symptoms are slight or altogether wanting. If, however, a careful inspection of the interior of the nares is made, rather large quantities of mucus may be seen in the region of the infundibulum, while the lower portion of the nasal chambers is comparatively free from it.

It is not so easy to diagnose catarrhal inflammations of the posterior ethmoidal and sphenoidal cells, as the examination must be made with a postrhinoscopic mirror. While it may be difficult or impossible to diagnose catarrhal sinusitis, the possible etiologic relation of such a condition should not be lost sight of.

Secondary Effects of Rhinitis or Nasal Obstructions.

These are much more serious than is usually supposed.

Most **catarrhal inflammations of the lower respiratory tract** are largely due to morbid conditions within the *nose* or postnasal space. The continued inhalation of improperly prepared air affects the vaso-motor system of the lower respiratory tract in such a way that the blood-supply is unstable or erratic. Recurrent hyperæmia and subacute inflammation of the larynx and bronchi are thus favored. Catarrhal diseases of the lower respiratory air-tract should always lead the physician to examine the *nose* and nasopharyngeal space for pathologic conditions.

Lymphoid enlargement in children: It may be laid down as a general law that "slight local irritation is likely to cause hypertrophy of the lymphoid tissue in children." Thus the faucial and pharyngeal tonsils often enlarge after an attack of one of the exanthematous fevers. So slight an irritation as an acute coryza may cause it. An immense amount of mischief might be prevented by the judicious use of sprays and douches in the course of the exanthematous fevers.

Acute Rhinitis.

Definition and varieties: This is an acute inflammation of the nasal mucous membrane; and it may be catarrhal; dry; croupous or fibrinous; diphtheritic; phlegmonous, or hyperæsthetic in type.

Acute Catarrhal Rhinitis; Acute Coryza; "Cold in the Head."

Etiology: (*a*) It frequently results from chill or exposure, especially if the feet are not well protected. (*b*) It may be epidemic during the spring or autumn in temperate climates.

(*c*) The exanthematous fevers are usually attended by an acute coryza, which is due to disturbances created within the system by the presence of the specific microörganism of the disease. (*d*) The inhalation of vapors and hot dust-laden air may excite it. (*e*) Hajek has isolated the Diplococcus coryzæ, but it is doubtful if it is a cause of coryza. (*f*) Chronic rhinitis and nasal stenosis are very prominent and frequent predisposing causes. Indeed, frequent attacks of acute coryza are almost pathognomonic signs of some form of chronic nasal disease. It has been held that repeated attacks of acute rhinitis are the cause of chronic rhinitis, but the reverse is more nearly correct.

(*g*) Tuberculosis and syphilis also act as predisposing dyscrasiæ.

Acute catarrhal rhinitis—symptoms: The *onset* is characterized by a feeling of chilliness without rigors, lassitude, nasal obstruction, and sneezing. This is soon followed by *serous discharge* from the nose, which is quite irritating to the skin of the anterior nares and upper lip. After two or three days the discharge becomes *mucopurulent*. It may remain in this condition for a few days or weeks. The *temperature* and *pulse* show a slight febrile movement. There is *frontal headache*, due to closure of the infundibula leading from the frontal sinuses. The air thus imprisoned within the sinuses is gradually absorbed, thus producing a partial vacuum, which in turn induces engorgement of the vessels of the lining mucosa.

Acute catarrhal rhinitis—treatment: (*a*) A few drops of spirits of camphor on loaf-sugar, taken every two or three hours, is a remedy of some value during the *early stages* of the attack. (*b*) A few drops of a 10 per cent. solution of menthol in chloroform rubbed in the palms of the hands and inhaled vigorously affords immediate relief to the nasal stenosis. (*c*) A snuff composed of the hydrochlorate of morphine, gr. ij; bismuth subnitrate, ʒiv; and pulverized acacia, ʒij, gives temporary relief to the nasal stenosis, modifies the intensity of the disease, and perhaps cuts short its duration. (*d*) Ten grains of quinine and five grains of Dover's powder taken at bedtime, with hot lemonade, are perhaps the most reliable remedy if given within the first twelve or twenty-four hours of the attack. (*e*) A capsule containing pulverized camphor, gr. $\frac{1}{2}$; extract of belladonna, gr. $\frac{1}{8}$; and bromide of quinine, gr. j, given every hour for three or four doses; or until the physiologic dryness of the throat occurs, is also a valuable remedy in the early stage. (*f*) The administration of a *saline cathartic* favors a speedy termination of the process by lowering the blood-tension, and by removing possible sources of irritation which may reside within the intestine.

In the *second stage*, or stage of profuse secretion, but little can be done to arrest the progress of the inflammation. The nose should, however, be cleared of secretion by the use of an alkaline solution (Dobel's) used with a nasal douche. Eight grains of the chloride of sodium to the ounce of tepid milk are also a very grateful wash and diluent in this stage. The washes may be followed by inhalation of the hot vapor of the compound tincture of benzoin, ℥iv; the oil of tar, ʒj; in one-half pint of boiling water. Oily sprays containing a small amount of the oil of eucalyptus and menthol are valuable protective applications which may be used after thorough cleansing of the nares.

Should there be great engorgement of the nasal mucous membrane, the openings into the accessory sinuses may be closed. When this is the case there is danger of *acute sinusitis*. Treatment should be promptly directed to this condition, hoping thereby to abort the threatened invasion. This may be done by first cleansing the nose by the use of Seiler's or Dobel's solution, after which the parts should be thoroughly cocainized. After the nasal engorgement is thus reduced, a 10 per cent. solution of antipyrine should be applied to prolong the period of arterial contraction.

These applications should be repeated every three or four hours. The application of a solution of the *extract of suprarenal capsule* may be used to contract the vessels in those cases characterized by extreme engorgement. Indeed, this remedy seems to promise the most satisfactory results. It should be freshly prepared, as it rapidly undergoes bacterial decomposition and may give rise to infection.

A moderately *light diet* and slightly increased weight of underclothing will aid materially in shortening the course of the disease.

Acute catarrhal rhinitis—prophylactic treatment: The prevention of repeated attacks of coryza must be based upon two recognized facts, namely: coryza is due (*a*) to a preëxisting chronic rhinitis or other nasal disease; and (*b*) to an unstable condition of the vaso-motor nervous system.

Prophylactic treatment based upon the first of these facts will be addressed to the cure of the preëxisting chronic nasal catarrh or obstruction.

That based upon the *second proposition* will embrace such measures as will restore tonicity to the vaso-motor system. This can be best and most simply accomplished by regular exercise in the open air, and cold sponge-baths on rising, followed by friction of the skin. The skin is the largest glandular structure in the body, and has the most extensive capillary circulation, hence diminished activity of its functions is attended by auto-intoxication, which disturbs the vaso-motor system. In other words, if the functional activity of the skin is maintained, the tendency to take cold will be very much diminished.

Acute Dry Rhinitis.

Symptoms: This is a modified form of the acute catarrhal type in which there is little or no secretion, although the turgescence progresses until there is stenosis. There are an intense diffused headache and pain across the bridge of the nose and back of the eyes, due to turgescence of the mucous membrane lining the ethmoidal and sphenoidal sinuses. Objectively the middle turbinals are swollen, dry, and glazed.

Acute dry rhinitis—treatment: It should be about the same as in the catarrhal variety. Hot fomentations across the bridge of the nose and over the eyes afford great relief to the pain.

Acute Croupous or Fibrinous Rhinitis.

Diagnosis and pathology: This should not be confounded with *diphtheritic rhinitis*, in which the Kleb's-Löffler bacillus is found. All membranous formations in the nose, however, should arouse suspicion of diphtheria, and most careful bacteriologic examination be made. The membrane is inflammatory in origin and is loosely attached to the mucous membrane, while in true diptheria it is firmly attached. Cocaine will neither anæsthetize nor reduce the turgescence.

Treatment: The administration of iron and local alkaline washes is indicated. The false membrane should be removed daily.

Diphtheritic Rhinitis.

Occurrence: It may precede, follow, or appear at the same time, as the faucial diphtheria.

The **treatment** should be as for diphtheria in the lower air-tract, the nose being frequently irrigated with boric-acid or other (alkaline) solution.

Acute Phlegmonous Rhinitis. (Abscess of the Septum.)

Occurrence and symptoms: This may exist as furuncles near the nasal orifice; or, more rarely, as abscess of the septum.

Abscess of the septum is usually the result of traumatism.

At first the swelling is hard, but in a few days it becomes soft and fluctuating to the touch. It is attended by local pain and redness, while the temperature-curve may be slightly upward.

Acute phlegmonous rhinitis—treatment: Before pus formation the local application of a 5 per cent. solution of carbolic acid, or of the tincture of iodine and ice, may check the process. After pus formation free incision and drainage will usually be successful. In strumous children, and in the more chronic cases, it may be necessary to curette the abscess cavity and swab it with the tincture of iodine.

Specific Forms of Rhinitis.

Syphilitic and **tubercular rhinitis** will be considered in connection with respectively *syphilis* and *tuberculosis of the nose.*

Rhinitis of specific fevers: Most of the exanthematous fevers are attended by acute rhinitis, characterized by the peculiar eruption of the disease. The nares should be frequently douched with antiseptic alkaline solutions.

Hyperæsthetic rhinitis: *Hay fever*, or hyperæsthetic rhinitis, will be described later in connection with the *neuroses.*

Glanders.

Occurrence: This disease is rare in man, but may be transmitted from the horse, in which it is common. It is a highly contagious disease, characterized by submucous infiltrations, or granulation tumors, which undergo rapid degeneration and discharge a foul, offensive material. The disease is essentially an acute one, but may become chronic.

Etiology: The Löffler Bacillus mallei is the specific organism which causes the disease. Horsemen or stablemen who spend much of their time with horses are especially liable to become infected. Abrasions of the mucous surface add to the probability of infection.

Glanders—symptoms: The *acute form* or stage of the disease presents much the same clinical picture as that of most acute infectious fevers. Chills, rigors, lassitude, headache, nausea, vomiting, dyspnœa, and diarrhœa are usually present. Local heat, redness, swelling, and pain are present. The adjacent lymphatic glands become swollen and painful. Translucent nodules, which finally ulcerate, form on the surface of the mucous membrane and discharge a foul-smelling fluid. The pulse and temperature run high, and there are a profound toxæmia, exhaustion, and profuse sweating, which finally terminate in death. The close of the disease very much resembles that of typhoid fever.

The *chronic form* runs a less rapid and milder course. The symptoms are much the same as in the acute form, with their intensity much diminished.

Glanders—treatment: The toxæmia and exhaustion are somewhat modified by the administration of quinine, iron, strychnine, and whiskey. Abscesses formed in the nasal or adjacent tissues should be promptly opened, their walls cauterized and cleansed frequently with antiseptic washes. Potassium iodide will modify the course of the disease in some cases.

Rhinoscleroma.

A very rare disease, **characterized** by the formation of firm nodular masses in the submucosa; although it may extend to the skin of the nose and adjacent structures. The disease progresses slowly by extension, is painless, and does not show a tendency to ulcerate. The nodules are composed of fibrous tissue and small round cells. There are no constitutional symptoms, although it is probably due to a microörganism.

The **etiology** is extremely obscure. It is probably due to the *Bacillus rhinoscleroma*, an encapsulated, short, rod-like germ with rounded ends. Sex, age, and local nasal conditions are not known to exert a marked influence, although it is most common in southeastern Europe, or at least has been most often described there.

Rhinoscleroma—symptoms: The infiltration nodules or plaques usually begin at the margins of the nares at the union of the skin and mucous membrane, and are firm; the skin over them is smooth, slightly shiny, hairless, and very slightly congested from venous stasis. There are no discharge, ulceration, or pain. The infiltration may extend to the skin of the external nose and face, but usually extends backward into the nares, or to the larynx in very rare instances. The nose becomes misshapen and stiff, and finally more or less stenosed. This changed appearance of the nose and the compensatory mouth-breathing give the patient quite a peculiar physiognomy. Laryngeal spasms and dyspnœa may occur when the disease has advanced to the laryngeal submucosa. The uvula may become involved and subsequently atrophied.

Rhinoscleroma—treatment: Such constitutional remedies as mercury and the iodides have been tried, but with little if any effect. Total removal of the tissue is followed by its return. The only point to be gained by surgical interference is the relief of the nasal stenosis and its attendant sequelæ.

Chronic Rhinitis.

Classification: The various types and manifestations of chronic rhinitis will be better understood after a study of a classification based upon the pathology of the different types. The excellent classification given by MacDonald is given below:

A. Chronic catarrhal rhinitis.
    1. As affecting the erectile tissue:
        a. Chronic catarrhal rhinitis with vascular tumefaction of the erectile tissue.

        - *b.* Chronic catarrhal rhinitis with collapse of the erectile tissue.
        - *c.* Chronic catarrhal rhinitis with true hypertrophy and œdema of the erectile tissue.
    - 2. As affecting the ethmoid bone:
        - *a.* Hyperplasia of the middle turbinated body.
        - *b.* Caries and exfoliation of the bone with suppuration, which may involve the accessory sinuses.
        - *c.* Mucous polypus and cysts as a consequence and concomitant of *a* and *b*.
- B. Chronic dry rhinitis.
    - 1. With simple mucous secretion.
    - 2. With mucopurulent secretion, commonly called ozæna; and in the later stages correctly styled atrophic rhinitis.

This classification, while perhaps not sufficiently subdivided to include all cases of *chronic rhinitis*, is, nevertheless, based upon broad physiologic and pathologic principles, and embraces all the essential features of the various manifestations of chronic rhinitis. It is well to study inflammations affecting the erectile tissues in a class separate from inflammations affecting the ethmoid bone, as in the former the clinical and pathologic phenomena are quite different from those in the latter.

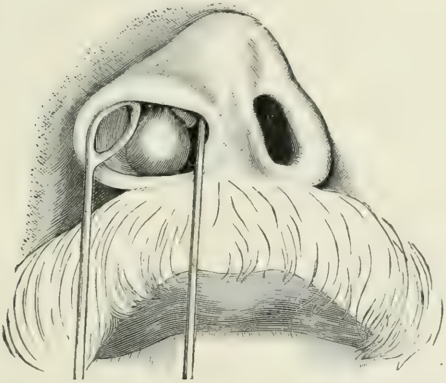
While *dry rhinitis* may be a later and infected stage of simple catarrhal rhinitis, the clinical phenomena and the pathologic changes are so marked as to warrant a separate consideration.

Chronic Catarrhal Rhinitis with Vascular Tumefaction.

Pathology: It will be recalled that along the free border of the inferior turbinated body, and its posterior and anterior ends, are located "swell bodies" which have the power to become tumefied or engorged with blood, and to collapse under certain physiologic stimuli. These are the erectile tissue of the nose. Their function is to transude serum and warm the inspired air. Under certain conditions the tumefaction may be more or less persistent, or present periods of intermission.

The patient complains that at times one nostril (Fig. 84) is closed while the other is open, and *vice versâ*. Or he may state that when lying upon the right side the right nostril is closed, and upon turning to the left side the order is reversed. These are the most characteristic phenomena of chronic rhinitis with tumefaction of the erectile tissue.

FIG. 84.



Dilated nostril, showing anterior tumefaction. (Seiler.)

Chronic catarrhal rhinitis with tumefaction—etiology: Races of people or individuals who have prominent, narrow noses are more liable to catarrhal affections than those who have wider nasal chambers. Hence the Hebrew race are very commonly affected with chronic rhinitis, while negroes are rarely thus affected. The state of the general health does not seem to influence materially, or at least to predispose to, catarrhal rhinitis. It is quite common in those accredited with good health, in whom it occurs quite as often or oftener than in those in poor health. Climate seems to exert a marked influence in its promotion, as it is most common in temperate zones, and especially on the great bodies of water where the atmosphere is subjected to frequent and radical changes in temperature and humidity. It is less common in places or countries having a uniformly moist and warm atmosphere, and in those countries where the atmosphere is uniformly dry and cold. It is more prevalent in males, perhaps on account of their greater exposure to the inclemencies of the weather. Age exerts an important influence, as catarrhal inflammations occur chiefly during the period of adolescence and early adult life. It may occur, however, at any period of life. Certain occupations which necessitate the inhalation of dust-laden air or irritating vapors predispose to it. Frequent attacks of acute coryza were formerly thought to be the chief cause of chronic rhinitis. While such attacks do undoubtedly aggravate existing chronic rhinitis, it can hardly be held that they are the chief cause of it. It is more nearly correct to say that chronic rhinitis predisposes to frequent attacks of acute coryza. Indeed, one may almost certainly predict that those suffering from repeated attacks of acute rhinitis are suffering from some form of chronic rhinitis. Undoubtedly the most prolific source of chronic inflammations of the Schneiderian membrane is anterior nasal obstruction. As has been already explained under the General Etiology of Rhinitis, anterior nasal obstruction is attended by rarefaction of the air in the nasal chambers posterior to the obstruction. This leads to chronic engorgement or tumefaction of the erectile tissue, which, in turn, still further adds to the conditions which give rise to the rarefaction. Vasomotor control of the erectile tissue is in time impaired or lost, and the clinical phenomenon known as "alternating stenosis" is established.

The *conditions which give rise to anterior nasal obstruction* are a narrow nasal chamber, flat or indrawn nasal alæ, spurs, ridges, and deflections of the septum. An acute attack of rhinitis occasions anterior nasal stenosis of temporary duration, which leads to impairment of the vaso-motor control, and thus may leave a condition which may develop into chronic rhinitis.

Chronic rhinitis with tumefaction—symptoms: The most prominent subjective symptom is alternating or fugitive nasal stenosis attended by a more or less profuse mucous discharge, which becomes mucopurulent when an attack of acute rhinitis supervenes upon the chronic form. The patient will "sniffle" and "snuff," or blow his nose, attempting thereby to free it from the sensation of stuffiness and obstruction. While the general health may be good, he belongs to the class known as neurotics. Repeated attacks of cold in the head occur, which may persist for many weeks.

Objectively, the inferior turbinate body will appear as a rounded purplish-red tumor almost filling the inferior meatus, and oftentimes touching or nearly touching the septum. If not touching the septum, strings of ropy mucus may be seen extending from it to the swollen turbinate body. Upon pressure with a probe the turgid inferior turbinate pits, thereby demonstrating its fluid contents, and proving the absence of true hypertrophy. This may also be demonstrated by swabbing the nose with a 4 per cent. solution of cocaine, which after two or three minutes will cause the erectile tissue to collapse firmly over the inferior turbinal.

On account of the chronic over-nutrition of the parts, there is a tendency for this form of catarrh to merge gradually into "chronic rhinitis with true hypertrophy." Indeed, it may be truly regarded as an *early stage* of *hypertrophic rhinitis.*

Prognosis: If the cause of the anterior nasal stenosis is removed, a cure, or at least an almost complete relief, of the distressing symptoms will follow.

Chronic rhinitis with tumefaction—treatment: The treatment is topical and surgical. The *topical* treatment consists of alkaline and antiseptic sprays, as Seiler's or Dobel's solution, used during the morning toilet, and just before retiring at night. After thus cleansing the nose the patient should be directed to use a spray containing 10 per cent. of the oil of eucalyptus; 5 per cent. of the oil of pine-needles; and 1 per cent. of menthol in liquid alboline. The treatment at the office should consist of local massage of the turgid mucous membrane. This may be accomplished by means of a small silver probe, upon the end of which a small piece of cotton about three times the size of a wheat-grain is engaged. This is dipped in a 10 per cent. ichthyol ointment and rubbed on the swollen inferior turbinal with a gentle but rapid to-and-fro wrist movement. The probe should be held lightly with the tips of the thumb and second finger, care being taken to avoid the use of a third finger, as this renders the manipulation of the probe so stiff and awkward that the mucous membrane is liable to be seriously bruised or otherwise injured.

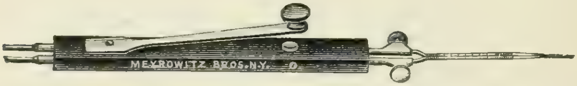
Intranasal massage is of value for three reasons: (*a*) by it the vascular tumefaction is temporarily overcome, fresh blood replacing it after a time; (*b*) the lymphatics are also unloaded, new lymph taking the place of the old, thereby establishing a more normal state of nutrition; and (*c*) the vaso-motor system is stimulated to greater tonicity, and therefore has better control of the erectile tissue.

The *surgical treatment* consists of measures for the relief of the anterior nasal stenosis and for the reduction of the tumefaction of the erectile tissue. The methods of correcting anterior nasal stenosis are described elsewhere under *disease* and *deformities of the nasal septum.*

There are two methods of *reducing the erectile tissue*, namely: (*a*) superficial linear cauterization; (*b*) deep incisions with a knife, including the whole length of the erectile tissue of the inferior turbinated.

Superficial linear cauterization should be done as follows: Cocainize about one inch of the surface anteroposteriorly with a 4 per cent. solution of cocaine applied by means of a thin film of cotton over the area to be cauterized. After a period of from five to eight minutes the cotton should be removed, when collapse and complete anæsthesia will have taken place. The cauterization is best done by the use of a straight cautery knife or electrode (Fig. 85) applied cold to the surface of greatest convexity, the electrode being moved with a sliding motion backward and forward. While the electrode is thus in motion, the electric current should be connected, and a superficial linear burn produced. The electrode should be kept in constant motion until the current is disconnected, as otherwise the eschar will adhere to the hot wire as it is removed. As the burn does not extend to the submucous tissue, but little cicatricial contraction follows its use. It seems, however, to exert a powerful influence upon the vaso-motor control.

FIG. 85.



Cautery handle and electrode.

Four days later the opposite side may be likewise treated, thus alternating until the whole length of both turbinals has been cauterized. A 2 per cent. oily solution of cam-menthol sprayed into the nostril will prevent inflammatory reaction.

Deep linear incisions: These are made along the whole length of the erectile tissue of the inferior turbinated body. Two such incisions running parallel may be made at one sitting. The parts should be first cocainized as for cauterization. Profuse hemorrhage follows, but ceases after a few seconds or minutes. If after cocainization an application of a solution of the suprarenal capsule extract is applied, the operation will be bloodless.

The nose should be sprayed with the oily solution prescribed for home treatment, after cauterization and linear incision, and a light piece of cotton inserted into the anterior nares to prevent the entrance of dust and germs.

Chronic Catarrhal Rhinitis with Collapse of the Erectile Tissue.

Etiology: It is doubtful if this condition should be classed as a true rhinitis. It is a symptom most often found in anæmic women, and tends to a spontaneous recovery when the anæmia is cured. The condition is sometimes seen in gouty individuals, although this is rare.

Chronic rhinitis with collapse—symptoms: These are mainly due to the loss of the respiratory functions of the nose, such as dryness of the nasal and pharyngeal mucous membranes. There is also more or less laryngitis present.

Upon *rhinoscopic examination* the inferior turbinated body will be seen as a long ridge projecting from the outer wall of the nose, over which the mucous membrane is drawn quite closely. At first view it will strike the observer that true atrophy is present. The mucous membrane is dry, glazed, and anæmic, with little or no incrustation. The condition is always bilateral. If crusts are present, they will be found upon the anterior third of the middle turbinal. The sense of smell is not affected. Ulceration is never present.

Chronic rhinitis with collapse—treatment: But little can be said in this connection. It should be directed to the removal of the causes producing the anæmia. Local antiseptic washes and massage, as already described, are sometimes followed by engorgement of the collapsed tissues.

Chronic Catarrhal Rhinitis with True Hypertrophy (Hypertrophic Rhinitis).

Definition: This is a chronic inflammation of the nasal mucosa characterized by the presence of hypertrophy and œdema of the erectile tissue. The etiology has already been given under chronic catarrhal rhinitis with tumefaction of the erectile tissue. The hypertrophic variety is but a more advanced stage of rhinitis with tumefaction. Indeed, the two types are often associated in the same nose, portions of the membrane having undergone hypertrophy while other portions remain in the state of turgescence.

Hypertrophic rhinitis—symptoms: The symptoms are identical with those of rhinitis with tumefaction, with the exception that the stenosis is more constant and the secretions less fluid. *Objectively*, the inferior turbinated body appears as a more or less nodular mass, which pits but slightly upon pressure and does not collapse markedly upon the application of cocaine. These points of difference stand out in sharp contrast to those presented in the turgescent variety.

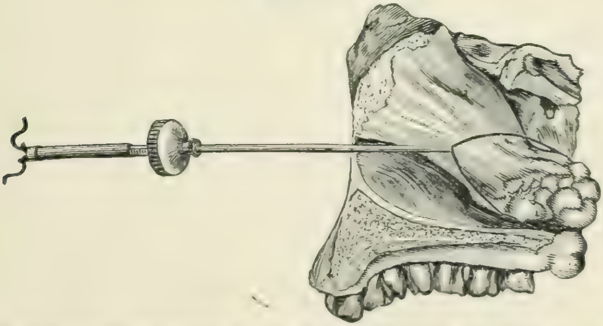
Postrhinoscopic examination often reveals a bluish nodular conformation of the end of the inferior turbinals. They have been described as having a "mulberry appearance." As the tissue in this locality is chiefly vascular in structure, the hypertrophy takes the form of a varicosity. In other words, "the mulberry appearance" is due to enlargement or hypertrophy of the veins composing the erectile tissue. The mucosa in this locality being very thin, the bluish color of the venous blood is apparent.

Cough is sometimes present, and is due to intranasal pressure and to the presence of pharyngitis and bronchitis, which are the result of impaired nasal respiration. Postnasal catarrh is often present, more especially when the posterior ends of the inferior turbinals present the "mulberry" enlargements. Laryngitis may or may not be present.

Hypertrophic rhinitis—treatment: Home treatment, as directed for chronic catarrhal rhinitis with tumefaction of the erectile tissue, should be carried on. The office treatment should be directed toward the removal of the causes of anterior nasal stenosis and the reduction of the hypertrophic tissue. For the former, all spurs, ridges, exostoses, and septal deflections should receive appropriate surgical treatment as described under diseases and deformities of the nasal septum. If there is collapse of the alæ nasi, suitable silver tubes should be worn until the anterior nasal orifices are larger and no longer obstruct respiration. If there is dislocation of the columnar cartilage, it should be resected and the wound closed with sutures.

The hypertrophied erectile tissue may be reduced in bulk or removed *en masse*. If the amount of hypertrophy is small and combined with tumefaction from engorgement of the erectile tissue and interstitial œdema, deep cauterization may afford enough reduction to overcome the nasal obstruction. This is done as described under chronic rhinitis with collapse, except the linear cauterization is deeper. The submucous tissue is included in the operation; hence cicatricial contraction will follow. If the hypertrophy is large and causes marked nasal obstruction, it should be removed with the cold-wire snare or file-edged scissors. The posterior "mulberry" hypertrophy should be removed with a cold-wire snare (Figs. 86–89), introduced through the anterior nares and insinuated over

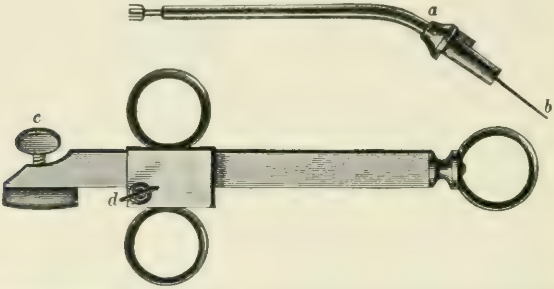
FIG. 86.



Jarvis's snare in position, showing loop around a posterior hypertrophy. (Jarvis.)

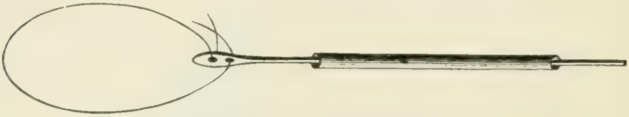
the growth with the aid of the postrhinoscopic mirror or the finger introduced through the mouth. It should be tightened slowly, so as to prevent the occurrence of severe hemorrhage. At least a half hour should be consumed in the operation. A thin nasal splint made of soft or vulcanized rubber, introduced after cauterization, will prevent adhesions. It should be worn several hours a day for a week or more. The use of the splint may be dispensed with if the patient can be inspected every forty-eight hours.

FIG. 87.



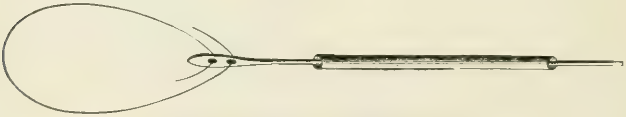
Schroetter snare: *a*, canula; *b*, stylet; *c*, binding-screw for canula; *d*, binding-screw for stylet.

FIG. 88.



Wire threaded for a vertical loop.

FIG. 89.



Wire threaded for a horizontal loop.

Such a course of treatment is usually attended with excellent results, as not only the cause but also the results of nasal stenosis are overcome. The glandular function of the mucosa is somewhat impaired; hence the secretions will be thicker than normal.

Chronic Catarrhal Rhinitis with Hyperplasia of the Middle Turbinated Body.

Pathological considerations: In true hypertrophy there is an increase of all the elements composing the normal tissue, while in hyperplasia there is an increase in one of the elements, usually the connective tissue. So the term hyperplasia as here used means a connective-tissue or fibrous thickening of the mucosa, while the ethmoid bone (which belongs to the connective-tissue group) is also enlarged. For reasons not well understood, there is a tendency to caries of the bone and polypoid degeneration of the hyperplastic membrane. It is probably due to obstructed drainage from the ethmoid cells. Ulceration occurs within them, the bone beneath becomes carious, is exfoliated, and granulations and polypi form. This explanation does not, however, adequately explain all cases. It would more nearly apply to those cases of polypi which are not true myxomata, but are œdematous granulation-tissue. The changed character of the secretion in the ethmoid cells, together with its retention and absorption, perhaps produces an irritation and disturbance of trophic processes which lead to myxomatous new formations.

My experience leads me to suspect that in the simple or uncomplicated form of hyperplasia there often coexists a simple catarrhal sinusitis or ethmoiditis. At a later period, when caries and exfoliation occur, there is often a suppurative sinusitis associated with it.

Hyperplastic rhinitis—symptoms: In simple hyperplasia the symptoms are not pronounced. They are chiefly referable to intranasal pressure from the encroachment of the middle turbinals upon the nasal bones and septum. This "squeezing in" process gives rise to neuralgic pains and a sense of fulness across the bridge of the nose, which may be somewhat widened and slightly sensitive to touch or pressure.

There is a catarrhal secretion from the mucous membrane which is usually not profuse, but shows a tendency to become inspissated, especially upon the anterior end of the middle turbinal. There may or may not be an associated hypertrophic rhinitis. Upon inspection the middle turbinal is seen to be large and redder than normal, the anterior end being covered with a film of inspissated mucus. It lies against the septum, which is often crowded to the opposite side. One or both sides may be involved. Sometimes one side is slightly hyperplastic while the other is markedly so.

Hyperplastic rhinitis—treatment: If there are no marked symptoms of pressure, surgical interference is to be deprecated. If, however, there are pressure-symptoms, the enlarged portion of the middle turbinal should be removed to relieve the pressure and afford better drainage to the ethmoid cells and the mucosa of the upper portion of the nose. This may be done with a wire snare, biting-forceps, or curved scissors.

Curved scissors, as modified by Holmes, afford the best results, as they do not crush the bone nor leave spiculæ to be exfoliated, as often happens after the use of the snare. The Grünwald punch-forceps are objectionable, as the tissue is taken away piece by piece and spiculæ of bone often come away for weeks after the operation.

Chronic Rhinitis with Caries and Suppuration.

Pathology: As has been explained, this is a later stage of chronic rhinitis with hyperplasia. The enlarged middle turbinal is seen in the middle meatus as a dark purplish tumor, which when probed yields readily to pressure. A blunt, heavy silver probe should be used, a sharp, slender one penetrating the tissue too easily. After a little manipulation the probe can be insinuated through the granulation-mass and rough, granular bone felt beneath, a granular osteitis probably being present. The opposite nostril may be in the stage of simple hyperplasia.

Again the appearances may be quite different. The lower free border of the middle turbinated may present a pendulous flap of membrane which is movable with the probe. In rarer cases polypi may spring from the pendulous flap. Occasionally the granular and œdematous mass is cleft or perforated, thus forming the so-called *cleavage of the middle turbinal.* A probe can be passed through this cleft and rough bare bone demonstrated; or in some cases the probe will pass into the ethmoidal cells, the bone having been absorbed. Œdematous granulation-tissue or true polypus may be present also. The cleft is usually covered with a crust of dried mucopus, and may be unnoticed unless the crust is removed. I have observed that when the bone is removed it is light colored and does not have the appearance usually presented by carious bone. I have observed the same condition in the middle ear in connection with chronic suppuration. The bone seems to derive some nourishment, probably through the periosteum reflected in the small cells and canals so numerous in the bones of these regions.

Chronic rhinitis with caries and suppuration—symptoms: The subjective symptoms are those referable to pressure and nasal obstruction. The pressure-symptoms are a dull ache or pain across the bridge of the nose; and a widening of the bridge. The obstructive signs are impaired nasal respiration, which causes more or less complete mouth-breathing; the patient is usually quite nervous.

The *objective symptoms* are an enlarged middle turbinal which more or less fills the middle meatus and is covered with pus or mucopus, some of which may have dried upon the surface. The probe enters through the cleft if it is present, or is insinuated around the swollen mass and finally comes in contact with bare bone, which transmits a rough sensation to the fingers. If the swollen mass is squeezed, pus will ooze from its under surface into the middle meatus, or from above into the superior meatus, according to the location of the point of discharge. Polypi and granulation-tissue are present in some cases. The discharge may come from the ethmoidal cells or from the middle turbinal proper. There is a strong probability of associated ethmoidal disease in these cases.

Prognosis: Under proper treatment the prognosis is good in the majority of cases. Spontaneous cures rarely occur. The most thorough surgical and antiseptic treatment is sometimes followed by but slight improvement.

Rhinitis with caries—treatment: Carious bone and suppurating cavities can only be successfully treated by establishing free drainage and removing the carious tissue. The middle turbinal should be freely opened with a sharp spoon-curette and the morbid bone and other matter removed ; or the turbinated body should be removed in part or *en masse*, thereby opening the ethmoidal cells, which may be explored and opened still further if necessary. Free drainage is thus established and the morbid material removed. The parts are open to irrigation with antiseptic fluids and the insufflation of antiseptic powders.

The cold-wire snare may be used to remove large portions of the diseased middle turbinated body. The operation may be begun with the curved scissors, cutting from before backward. The snare loop is then slipped through the cut, and the remaining portion removed. After the main body of the middle turbinal has been removed the ethmoidal cells can be explored and their walls broken down with a large dental burr or sharp spoon-curette. This may be done at subsequent sittings, many of which are often necessary. Budding granulations should be touched with trichloracetic acid as often as they form. Antiseptic washes and powders should be used daily, although it is impossible to obtain asepsis in the nasal cavities.

The ethmoidal cells may be entered with Grünwald's sharp spoon from beneath the middle turbinated body, working upward and outward, care being exercised to avoid entering the orbital cavity.

This class of cases requires great patience, the period of treatment often covering a period of many months.

Chronic Rhinitis with Cysts and Abscesses of the Middle Turbinal.

Pathology: The cysts occupy the body of the middle turbinal, and vary in size from a pea to that of a hickory-nut ; their walls are usually composed of bone covered with mucous membrane, and their contents are mucus. The walls may, however, be calcareous and their contents pus. Granulations are not present inside the cysts, although they may be upon the outside. The contents may drain into the antrum of

Highmore and produce the characteristic signs of empyema of that cavity. Antrum disease should always excite suspicion of ethmoidal or frontal sinus disease.

Symptoms: They are those due to pressure and obstruction —*i. e.*, irritation, pain, wide nasal bridge, mouth-breathing, etc. Objectively it may be shown that there are adhesions between the diseased body and the septum.

Rhinitis with cysts and abscess—treatment: It is about the same as that described under chronic rhinitis with caries and suppuration. If there are adhesions, they should be overcome by cautery, knife, or scissors. After this has been done the snare should be applied and the cyst removed. The true diagnosis is not often made before the operation.

Chronic Dry Rhinitis.

Synonyms: Atrophic Rhinitis; Simple Mucous Rhinitis; Ozæna; Mucopurulent Rhinitis.

Etiology: These conditions can scarcely be called a disease, as they are the results of previous disease, except in those rare cases in which the atrophy is primary. There are three causes which seem to embrace most of the facts in the etiology. They are as follows:

a. A *simple atrophic process* which is not dependent upon some other local disease of the mucous membrane. Meissner holds that *ozæna* (see below) is due to (primitive) a broad, shallow nose and congenital development of pavement-epithelium instead of the columnar (*mucus-producing*) variety.

b. *Pressure necrosis from distention* of the bloodvessels, as in chronic engorgement of the erectile tissue. This is a cyanotic congestion, the general circulation participating in the sluggish venous flow. The mucosa covering the vessels is kept upon a constant stretch; and pressure-atrophy results as in red atrophy of the liver.

c. *Sclerotic atrophy* due to preëxisting inflammation in which connective-tissue cells or fibres are deposited. These after a time contract and cut off the blood-supply and choke down the glandular structure of the membrane. Thus atrophy is established and functional activity diminished or destroyed.

a. Simple Atrophic Rhinitis.

Simple atrophy may take place in the nasal mucous membrane as well as elsewhere in the body.

The **etiology** is not clear, and yet it is probable that it is due to the presence of some irritant in the blood, as in syphilis, tuberculosis, scrofula, etc. At any rate, the terminal nerve-endings are degenerated and nutrition modified.

The **treatment** should be addressed to the constitutional dyscrasiæ, upon the disappearance of which the atrophic and ozænic processes will improve or disappear.

b. Atrophic Rhinitis Due to Pressure (Cyanotic Engorgement).

Etiology: (*a*) There is some lesion of the heart, kidneys, liver, or lungs which causes a damming back of the venous blood upon the nasal mucous membrane as well as elsewhere in the body. (*b*) Organs thus affected do not eliminate the waste-products as rapidly as they should, and they are retained in the blood, where they act as irritants, exciting inflammatory reaction of a low grade. These two factors account for the phenomena known as pressure-atrophy as it occurs in the nasal mucosa.

Atrophic rhinitis due to pressure—symptoms: Although there is true atrophy, the membrane is congested to such a degree that there is nasal stenosis. The mucosa of the nose is boggy, purplish-red in color, and inflamed. The ozænic odor may be slight. There is an exudation from the engorged vessels, but it is not a true mucous secretion. The skin of the nose may be red. There is a sense of fulness across the bridge, and frontal headache is commonly present. The conjunctiva may be injected and attended by an overflow of tears.

Kyle refers to a case due to organic mitral lesion. I now have a case of this character in which the whole mucosa of the upper respiratory tract is cyanotic; the tonsils are enlarged and markedly blue from cyanotic congestion.

Prognosis: This will depend upon the curability of the lesion giving rise to the cyanotic congestion.

It is obvious that the **treatment** in many cases must be palliative only.

c. Atrophic Rhinitis Due to Sclerosis.

Etiology: All the causes given under the various types of catarrhal rhinitis may act as causes of this type of atrophy. The inflammation attending them is followed by a deposit of fibrous connective-tissue cells, which cut off the blood-supply and choke down the glandular tissue. The functional activity is gradually lost and the true mucous elements of the membrane finally disappear. The secretions become thick and inspissated. They dry upon the surface of the membrane, where, through some change or other, they develop the *ozænic odor.* Various theories have been advanced in explanation of the odor, but none of them is proved. Among them are the following, which are suggestive if not conclusive:

Theories as to the origin of the ozænic odor:

a. Simple decomposition of the mucopus.

b. Degenerative changes in which certain fatty acids are liberated which give rise to the odor.

c. The presence of certain bacteria, as the Bacillus fœtidus.

Ozæna as a symptom: Ozæna is not a disease, but a sign of certain diseased conditions. Its primary meaning is a "stench," and it is in this sense that it is here used. The fetid odor is associated with an inspissated secretion, which is seen in the form of greenish crusts covering the whole of the nasal mucous membrane. There are certain other peculiar conditions associated with it, especially in those cases in which there is marked atrophy of the mucosa. For instance, the nose is broad and flat, the tip somewhat elevated, and the blood anæmic. The anæmia is secondary and not primary as in chronic rhinitis with collapse of the erectile tissue. The absorption of septic material and the loss of the respiratory functions of the nose are probably the chief causes of the anæmia. It is a well-recognized fact that in obstruction from the presence of postnasal adenoids there is anæmia, which quickly disappears after their removal.

The ozæna of *syphilis* is somewhat different from that due to other causes. The odor is more offensive and the secretion richer in broken-down and necrotic *débris.* The mucous membrane becomes atrophic in the later stages, and after a longer period the secretion and foul odor may spontaneously disappear and leave a comparatively clean but sclerotic membrane.

The ozænic odor tends to disappear spontaneously after a number of years, hence is a self-limited symptom. The mucous membrane, however, is left very much damaged. Its histologic character and physiologic function are changed or entirely lost.

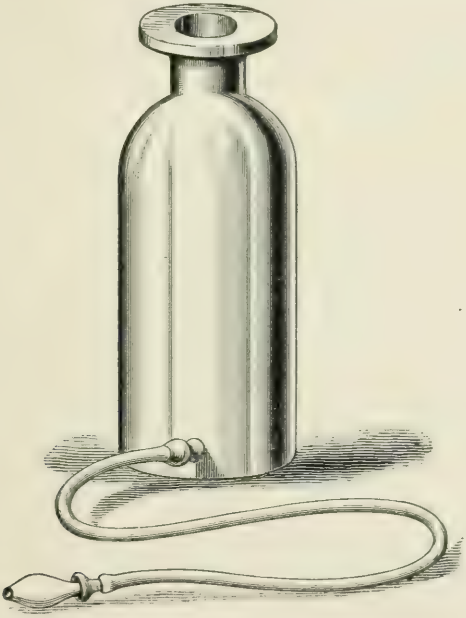
Atrophic rhinitis due to sclerosis—symptoms: The symptoms vary with the state of advancement and activity of the process. The clinical picture usually presents the features as seen in the comparative table given below. This is adapted from MacDonald's work on *Diseases of the Nose:*

Chronic Rhinitis with Collapse of the Erectile Tissue.	*Atrophic Rhinitis with Sclerosis and Mucopus Secretion. Ozæna.*
1. Chiefly in anæmic women. The anæmia is primary.	1. Chiefly in women and children; all subjects become anæmic. The anæmia is secondary to the ozæna.
2. No peculiarity of physiognomy.	2. Small, sunken, wide noses with wide nasal fossæ.
3. Mucous membrane anæmic.	3. Mucous membrane anæmic.
4. Collapse of erectile tissue; no tendency to atrophy.	4. Collapse of the erectile tissue; tendency to atrophy of the mucous membrane.
5. No ulceration.	5. No perceptible superficial ulcer, except from picking the nose.
6. Always bilateral.	6. Usually bilateral; may be unilateral.
7. Spontaneous cure if the anæmia is relieved.	7. After some years there is a tendency to improvement of symptoms. The ozænic symptoms disappear and the atrophy becomes more complete.
8. Olfaction not affected.	8. Olfaction is lost.
9. No characteristic odor.	9. Breath typically ozænic.
10. Little or no incrustation; if present, is limited to the anterior third of the middle turbinals.	10. Crusts equally distributed over every portion of the mucous membrane.

Atrophic rhinitis due to sclerosis—treatment: When seen in the early stage, the treatment should aim at (*a*) the removal of the causes of the inflammation that produce the sclerotic process; and (*b*) intranasal cleanliness.

a. The *causes* of the inflammation are numerous. Some have already been considered under acute catarrhal and infective rhinitis, chronic catarrhal rhinitis with tumefaction and turgescence of the erectile tissue; or with congenital primitive nose and pavement-epithelium. Others are traumatism, deflection, and other obstructive affections of the septum. By the removal of these exciting causes of the inflammation the sclerotic process is checked or stopped altogether.

Fig. 90.



Nasal douche.

b. *Intranasal cleanliness* is obtained by the use of antiseptic douches containing a liberal amount of mild alkalies in them to soften and dissolve the crusts and tenacious mucopus. A solution of 8 grains of sodium bicarbonate to the ounce of water as hot as can be borne should be forcibly injected into the nostrils at frequent intervals during the day. The Whitall-Tatum nasal douche (Fig. 90) is well adapted for this purpose. The patient should be instructed to clear the nose by blowing after each injection. The injection should be administered by the physician at first, as the patient will not or cannot thoroughly cleanse his nose. After freeing the nostrils from crusts and tenacious mucus a warm antiseptic aqueous solution of borax, sodium bicarbonate, oleum eucalypti, carbolic acid, glycerin, and alcohol should be injected into the nostrils. A two-ounce hard-rubber syringe is well adapted for this purpose, as considerable force is necessary to dislodge the crusts.

Sometimes is is expedient to introduce cotton-wool saturated with an aqueous or glycerin solution of ichthyol. After twenty to thirty minutes the crusts will be softened and detached from the mucous membrane. They are then easily removed by blowing, and the use of a cotton-wound probe. The nose should then be douched as already described. This course of treatment if faithfully carried out will bring about great relief in a large number of cases.

Mildly astringent stimulating solutions, or powders, are sometimes of value in overcoming the local pus infection. Powders with 5 to 20 per cent. of silver nitrate, or 1 : 2000 trichloracetic acid solution may be used for this purpose.

Associated sinus disease should be treated as described under *Accessory Sinuses.*

Syphilis of the Nose.

Primary infection of the nose is rare.

The **secondary symptoms** rarely show themselves, although mucous patches have been observed in the anterior nares.

The second stage may be manifested as an exanthema of the nasal mucous membrane, the patient seeming to suffer from an aggravated "cold in the head." In infants an obstinate catarrh should arouse suspicion of specific disease. In infantile cases there is profuse secretion which becomes dried, forming crusts in the nose. The bridge of the nose is flat.

The **tertiary stage** may come quite early, necrosis of bone having occurred at the seventh month after the primary infection. It usually occurs at from two to five years after infection.

The tertiary stage affecting the septum in adults is accompanied by little or no pain. If it affects the inferior turbinal, a boring pain is present.

The **treatment** should be about the same as for syphilis in other parts of the body. The following prescription should be used during the administration of mercury :

℞. Potassii chloratis,	gr. xc–cxx ;
Glycerin.,	ʒij ;
Aquæ,	ad ℥x.—M.

Sig. To be used as a mouth-wash in syphilis during the administration of mercury.

Infants with syphilitic coryza should be fed with a bottle, and the syrup of the iodide of iron, cod-liver oil, and mercury be given. The nostrils should be kept free from crusts and thick mucus by means of an alkaline spray.

Sequestra of necrosed bone should be carefully removed through the anterior nares. The horrible odor may be somewhat overcome by a douche of 2 per cent. carbolic lotion. Where there is extensive ulceration, employ calomel fumigation by means of an apparatus consisting of a porcelain tube in which some of the powder is heated over a spirit-lamp. In the meanwhile a gentle current of air should be forced through the tube with a hand bulb or compressed-air chamber. Where there is a deep ulceration the following prescription should be applied :

℞. Liq. hydrarg. nitratis,	ʒj–iv ;
Aquæ,	q. s. ad ʒj.—M.

Sig. To be applied to the sloughing ulcer of tertiary syphilis. As the application is very painful, cocaine should first be applied.

If in spite of the foregoing treatment the rapid destructive process continues, inunctions of mercury should be used. The excoriations which occur on the upper lip in syphilitic ozæna and in syphilitic coryza of infants should be frequently treated with an ointment containing 30 grains of zinc oxide and 2 grains of morphine acetate to the ounce of vaseline.

Tuberculosis of the Nose.

Occurrence: About twenty authentic cases of tuberculosis have been reported. The author has seen one such case affecting the cartilaginous portion of the septum. The condition is rarely primary. The author's case seemed to be primary, having existed for eight years without sign of tuberculosis in any other part of the body. The septum is the favorite location, although the inferior turbinal may be affected.

Diagnosis: Microscopic examination of the tissue for tubercle bacilli should be made.

In the author's case the microscopic examination was negative. Guinea-pig inoculations, however, developed tubercle bacilli in great abundance. During the warm summer months the ulcer would almost entirely heal. In November or December it would begin to spread, and was attended by slight pain. The appearance of the ulcer changed rapidly from time to time.

The tubercular ulcer is nodular and of a grayish color, while those of lupus are red and irritable in appearance. The ulcerative process is much slower than in syphilis. It is usually located upon the cartilaginous portion of the septum, while in syphilis the bony portion is affected.

Tuberculosis of the nose—treatment: This should be general and local. Creosote should be given, beginning with 3-drop doses three times a day, gradually increasing the dose to 10 or 15 minims or to the point of toleration. Cod-liver oil, iron, and the syrup of the hypophosphites should be given as for general tuberculosis.

The *local treatment* consists in curettement with a sharp spoon, followed by the application of a 50 per cent. solution of lactic acid. A 5 or 10 per cent. solution of menthol in olive oil may be used locally as a palliative measure.

Tuberculin is of very doubtful value.

In spite of all treatment there is a strong tendency toward a recurrence of the disease.

Lupus of the Nose.

The **etiology** of this affection is closely allied to tuberculosis of the nose, but has a somewhat different clinical aspect. Lupus of the mucous membrane of the nose is usually secondary to and associated with a similar affection of the skin of the face. The nodules are about the size of a pin-head, while those of tuberculosis are larger. The tubercular nodules tend to coalesce, while those of lupus have no such tendency. The nodules are red and irritable in appearance, while those of tuberculosis are grayish in color.

Treatment: Tonics and potassium iodid are of some value, but energetic local treatment is more effective. Curettement, followed by the application of the electric cautery, chromic, or lactic acid, is probably the most reliable method of procedure. The hypodermatic administration of tuberculin effects a temporary cure in some cases.

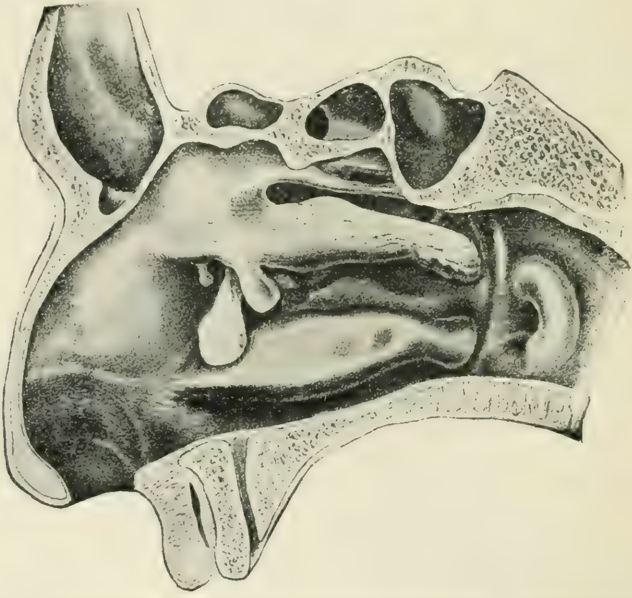
NASAL POLYPUS.

They are usually **located** on the free border of the middle turbinal or on the outer wall of the middle meatus about the lips of the hiatus semilunaris (Fig. 91). They rarely, if ever, grow from the septum or inferior turbinated body. The mucous membrane of the ethmoidal cells sometimes undergoes polypoid degeneration, which is attended by suppurative discharge and perforation of the middle turbinal walls. In these cases the polypi may grow entirely from the ethmoidal cells and protrude into the nasal cavity.

The **etiology** is but little understood. Various explanations have been offered, but none of them satisfactorily explains the origin of the growths. As they are often found with suppurative ethmoiditis, some have held that all nasal polypi are due to necrosing ethmoiditis. Still others have observed that nasal polypi are preceded by an œdematous condition of the middle turbinal, and have advanced the theory that they are the result of chronic œdema of a hyperplastic mucosa. They are more common in men than in women, and usually occur after the twentieth year (see, also, pp. 297 and 331).

Nasal polypus—symptoms: If the growths are small, the *subjective* symptoms may be slight. If they are numerous and are large enough to obstruct the respiratory tract of the nose, a pronounced nasal tone (diminished resonance) and mouth-breathing form the chief symptoms. The patient sometimes feels the growths flapping back and forth during respiration. If nasal stenosis is complete, great unrest and anxiety possess the patient. *Objectively* the growth appears as a pearly, grayish-blue translucent tumor, pendant from the region of the middle turbinal. It is freely movable with a probe.

Fig. 91.

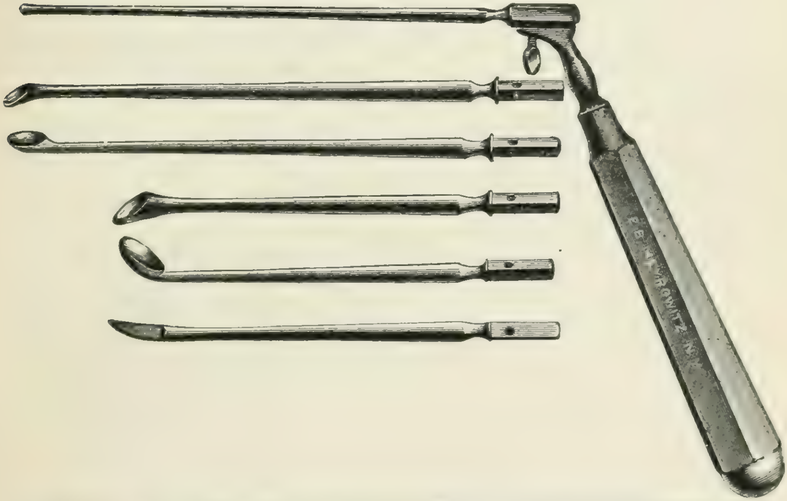


Outer wall of the right nasal cavity, exhibiting three polypi. (Zuckerkandl.)

Nasal polypus—treatment: Various local remedies have been suggested and tried for the purpose of causing the tumors to atrophy or slough away. Among these may be mentioned the submucous injection of a few minims of 10 per cent. solution of carbolic acid in glycerin; or a saturated solution of tannic acid. The author in his earlier experience used these remedies in cases in which there was but a single tumor with very good results. The blood-supply of the growth is thus cut off, the tumor sloughing away within a few days.

The *surgical removal* of the growths is so simple that it seems useless to resort to other methods of treatment. The cold- or hot-wire snare affords the most practical and effective method of treatment. After the application of a 5 per cent. solution of cocaine to the mucous membrane of the middle turbinated body and middle meatus, the wire loop should be insinuated around the growth as near its point of attachment as possible. The loop should then be gradually tightened and the growth thus removed. The base of the growth should be cauterized immediately after the operation, or within a few days, to prevent recurrence. If the hot wire is used, cauterization is accomplished at the time of the

Fig. 92.



Grünwald set, consisting of a probe, four curettes, and a curved, sharp-pointed bistoury, each of which can be inserted into a common handle.

operation. Twisting off the growth with forceps is a barbarous and obsolete method of treatment which is mentioned only for the purpose of condemning it.

If the middle turbinate bone and ethmoid cells are diseased, a portion of the bone should be included in the loop and removed at the same time. The after-treatment should consist of antiseptic sprays and douches and the insufflation of aristol or nosophen until the reparative process is complete.

In some cases the foregoing methods are not sufficiently thorough to remove all the budding granulations and polypoid masses. The mucous membrane and possibly the ethmoid bone and cells should be curetted with Grünwald's sharp spoons, shown in Fig. 92. The operation is not simple, and should be undertaken only after a thorough familiarity with the regional anatomy of the parts is acquired. There is some danger of penetrating the orbit, and even the cranial cavity, as the ethmoidal walls are thin and in case of chronic suppuration very much softened or necrosed. To the experienced operator, who knows the limits and location of the ethmoidal cells, these precautions seem trivial; but to the beginner or to the inexperienced nasal surgeon they are of considerable importance.

NASAL ACCESSORY SINUSES.

Location: Pneumatic spaces lined with mucous membrane open into the nasal chambers. They are: (*a*) the antrum of Highmore, or maxillary sinus; (*b*) the frontal sinus; (*c*) the anterior and middle ethmoidal cells; (*d*) the posterior ethmoidal cells; and (*e*) the sphenoidal cells.

They communicate with the nasal chamber through small openings, and the mucous membrane lining them is continuous with and similar to that of the nose. The lachrymal duct may be spoken of as an accessory sinus, but will not be so treated in this work. It opens into the inferior meatus just under the anterior end of the inferior turbinated body. The antrum of Highmore, frontal sinus, and anterior and middle ethmoidal cells open into the middle meatus. The anterior ethmoidal cells and frontal sinus open directly into the infundibulum which drains into the middle meatus. The posterior ethmoidal cells and sphenoidal sinuses open into the superior meatus. These points are of practical value in differentiating between empyema involving the various sinuses. It is comparatively easy, and therefore rather common, to diagnose empyema of the antrum and frontal sinuses; but it is much more difficult to diagnose empyema of the ethmoidal and sphenoidal sinuses.

Acute Sinusitis.

Symptoms: There is supraorbital headache; pain in the globe of the eye; deafness, tinnitus, and earache are often present; the symptoms do not yield to the usual remedies for nervous headache, but they disappear spontaneously after some days; or empyema develops. If empyema develops, the pus usually discharges into the nasopharynx, and is often erroneously regarded as a nasopharyngeal catarrh. The external signs are not marked in any of the acute non-suppurative inflammations except in *frontal sinusitis*, in which the eyelids may be œdematous, and lachrymation and closure of the tear-duct are present. Rhinoscopic examination under cocaine, causing collapse of the mucosa, reveals a boggy red membrane, with ecchymotic spots. The facial expression is dull, listless, and fatigued. The forehead and cheeks often perspire freely. The temperature is slightly elevated.

The **prognosis** in general is good. It is modified by the following conditions: (*a*) As the natural course of the disease is about eight days, early treatment is essential to the best results. If not promptly relieved, it may become subacute or chronic, and correspondingly difficult of removal. (*b*) An acute inflammation limited to the antrum is usually mild and terminates favorably. (*c*) The posterior wall of the frontal sinus is thin and sometimes absent. Frontal sinusitis is therefore liable to be complicated by brain or meningeal abscess. (*d*) The ethmoid cells are in intimate connection with the cranial cavity and the orbit, and the danger of basilar meningitis should not be overlooked. (*e*) The sphenoidal sinuses are in intimate connection with the cavernous sinuses, and inflammation in this region may, therefore, be complicated by thrombosis of the sinus.

Acute sinusitis—treatment: This is about the same as for acute rhinitis. The remedies should be vigorously pushed, and every precaution taken to protect the patient from draughts and inclement weather.

Chronic Sinusitis.

Varieties: The chronic inflammation may be catarrhal, dropsical, purulent, or cystic, according to the stage, location, and origin of the disease. The *catarrhal* variety is but a continuation of the acute catarrhal type. The *dropsical* is a result of the lessened resistance of the bloodvessels. The *purulent* is a late stage of the mucous or dropsical in which the secretions and exudates have become infected. The *cystic* variety is rare, and its etiology but little understood.

Empyema of the Antrum.

Etiology: Authorities differ as to the most frequent cause of suppurative inflammation of the antrum. In the author's opinion it is usually some form of intranasal disease, which obstructs the ostium maxillare. Older authorities and some recent ones gives *caries* of the first and second molars the first place. Careful examination of the teeth and nose should be made in every case. If a carious root is the cause, it can usually be demonstrated by the application of hot and cold metals to the crown of the affected tooth, pain being thereby produced. Tapping the tooth will show tenderness if caries is present. If there is an abscess at the root, it will protrude beyond the other teeth, so that in closing the mouth the elongated tooth will be painful. If there is caries at the root, the crown will have a dull, lifeless appearance.

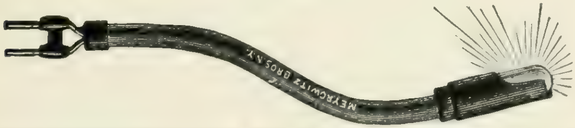
The conditions of the *nose* most liable to cause empyema of the antrum are polypi, hypertrophy of the middle turbinals, acute rhinitis, influenza, and suppurative disease of the frontal and anterior ethmoidal sinuses. Polypi growing from the middle turbinals and the lips of the hiatus semilunaris often obstruct the ostium maxillare. Add to this the swelling and infection attending an acute rhinitis and influenza, and the conditions are ideal for the development of antral disease. If there is suppuration of the frontal sinuses or anterior ethmoidal cells (both of which discharge into the infundibulum leading from the frontal sinuses and ending near the ostium maxillare), the pus may enter the antrum, which thus appears to be the primary seat of disease, while, in fact, it is but a receptacle for the pus discharged from above.

Empyema of the antrum—symptoms : If there is retention of pus, the signs of tension are present, viz., severe pain in the teeth of the upper jaw ; the osseous walls of the antrum are distended ; the abscess may point toward the mouth, nose, or orbit. If pointing toward the nose, the outer wall of the nose will bulge toward the septum. If toward the orbit, there will be more or less exophthalmos.

If drainage is free, there is unilateral discharge of pus from the nose. The patient complains of a chronic cold. The mucosa of the nose is sometimes swollen from the irritation resulting from the constant discharge of pus, and causes more or less nasal stenosis.

The patient complains of a bad smell (parosmia) or taste in the mouth. Upon inspecting the nostril the mucosa is red and swollen. Polypi growing from the middle turbinals and hypertrophy of the inferior turbinals may be present. The polypi may be a result of the irritation from the flow of pus through the ostium maxillare ; or they may be the cause of the suppurative inflammation. Yellow pus will be noticed in the region of the opening just under the anterior end of the middle turbinate body. After this is wiped away it may be seen to recur in a few minutes if the patient places his head between his knees, or inclines it toward the unaffected side.

FIG. 93.


MEYROWITZ BROS. N.Y.

Lamp for transillumination of the face for detecting disease of the antrum of Highmore.

When the diagnosis is in doubt the antrum should be aspirated by puncturing the outer wall of the nose with a curved trocar, or heavy hollow needle attached to a small syringe.

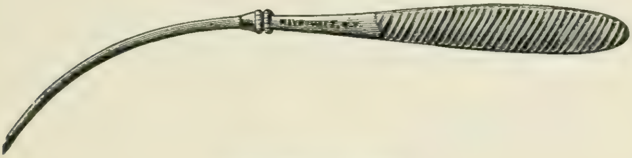
A five-candle power electric lamp (Fig. 93) placed in the mouth of the patient shows by transillumination the presence or absence of pus or other opaque matter in the antrum. The test should be made in a dark room, preferably one with walls painted black. The affected antrum will not transmit light so well as the one of the unaffected side, hence will appear darker and less luminous. Sometimes, owing to overdevelopment of the bony walls of one side there will be the same difference in transillumination. This fact should be borne in mind, otherwise an incorrect diagnosis may be made.

Antrum of Highmore—empyema—treatment: The treatment should be varied according to the etiology. If a carious tooth is the cause, it should be removed. If polypi or hypertrophy of the middle turbinated body cause it, they should receive appropriate surgical attention. If suppurative disease of the other accessory cavities is present, it should be cured before a favorable termination of the antral disease is possible.

In addition to the special surgical treatment just suggested, most cases require free drainage and lavage through an *artificial opening*. The best method of opening the antrum varies somewhat according to the peculiarities of the case. If a carious tooth or root is removed, the opening thus made can be utilized for drainage, the cavity being washed out once or twice daily through the opening. Warm antiseptic solutions of boric acid or other disinfectant should be used, the injected fluid flowing off through the nose. In obstinate cases a 4 per cent. solution of carbolic acid or a weak solution of iodine may be used. If the case is very obstinate, it will be necessary to explore the antrum for polypi and granulations.

In some cases the antrum is divided into several compartments by thin bony septa. It is often necessary to open the antrum with a dental burr or chisel through the canine fossa under a general anæsthetic, and break down the partitions before complete drainage and asepsis can be obtained. The opening should be large enough to afford a view of the interior and the introduction of a finger. In a case recently operated upon by the author the antrum was completely filled with polypi and granulation-tissue. All the walls of the antrum were thoroughly curetted with a stout Volkmann spoon and the cavity packed with sterilized gauze moistened in the compound tincture of benzoin. This dressing was not removed until the sixth day. On the sixth day the cavity was washed with warm boric-acid solution and more loosely repacked with gauze moistened in the same. The dressing was changed every two or three days thereafter and finally discontinued. The boric-acid lavage may need to be continued for several weeks.

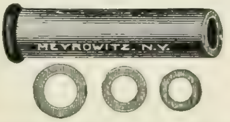
Fig. 94.



Myles trocar and canula.

Other methods of opening the antrum are through the outer walls of the nose just under the anterior end of the inferior turbinate with Myles trocar and canula (Fig. 94); through the alveolus, above and between the second bicuspid and first molar; or between the first and second bicuspids. Obturators made of gutta-percha, soft rubber, silver, or gold should be worn to prevent closure by granulation (Fig. 95).

Fig. 95.



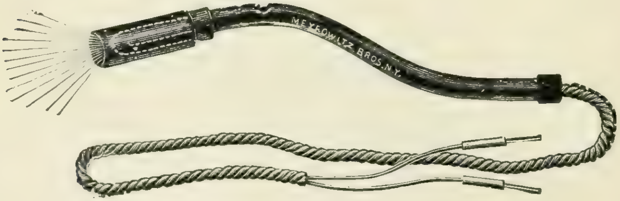
Myles soft-rubber drainage-tubes for the antrum. The actual length and the diameters of the three sizes are shown.

Frontal Sinus—Empyema.

Symptoms: It is usually associated with, or is secondary to, some other disease of the nose. The symptoms depend somewhat upon whether there is retention of inflammatory matter within the sinus, or whether there is free drainage of the same through the nose. With retention there are pain and tenderness upon palpation. The pus usually burrows toward the orbit, where it appears as a fluctuating tumor, causing diplopia, displacement of the eye, and epiphora. The sinus may be distended toward the cranial cavity, thereby causing vertigo, paralysis, and other cerebral symptoms. Should the cranial cavity be entered, meningitis, brain abscess, or phlebitis may occur and cause death unless prompt surgical relief is afforded.

Inflammation of the frontal sinus with free drainage through the nose gives rise to about the same signs and symptoms as suppurative inflammation of the antrum. It is often difficult to make a differential diagnosis of this type from antrum disease. The more frequent occurrence of antrum disease and a demonstration of its presence will, in doubtful cases, aid in arriving at a correct diagnosis. Sometimes there are swelling and tenderness in the frontal region, in which case the diagnosis is easy. The use of the electric lamp (Fig. 96) after the manner described under empyema of the antrum will help to clear the diagnosis. The lamp should be applied over the sinus and the transillumination noted.

FIG. 96.

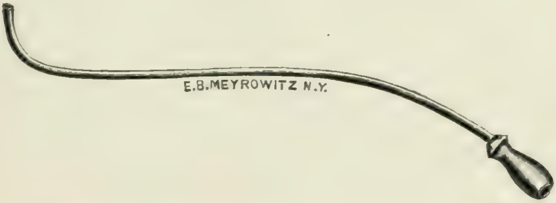

MEYROWITZ BROS. N.Y.

Lamp for transillumination of the frontal sinus.

Frontal sinus—empyema—treatment: In cases in which there is retention of the inflammatory products, the sinus should be opened from the outside at the point of greatest bulging or along the upper or lower margin of the eyebrow. The infundibulum should then be opened through the external opening thus made with a blunt-pointed probe or small spoon. The external wound should not be allowed to close until free drainage is established through the nose. The probe should be passed daily until the infundibulum remains patulous. The wound should be irrigated with antiseptic fluid (boric-acid solution) through a canula (Fig. 97) until a cure is established.

Where there is free drainage through the nose it is usually unnecessary to do the external operation. In some cases it is advisable to pass a stout probe between the septum and the anterior part of the middle turbinate body into the sinus, and enlarge the opening thus made with a small sharp spoon. The granulations which may tend to close the opening should be reduced by a bead of chromic acid fused upon the end of a probe. This method of operating is attended by considerable risk in the hands of one inexperienced in the surgery of the nose, and under any circumstances requires patience and perseverance. If mucous polypi or other morbid growths are found in the sinus, they should be removed by curettement.

FIG. 97.



Hartman canula for washing out the frontal sinus.

Ethmoidal Cells—Empyema.

Symptoms: The anterior ethmoidal cells open into the middle meatus through the infundibulum, while the posterior cells open into the superior meatus. An abscess in the ethmoidal cells may prove to be a very serious condition, and should receive the most prompt and efficient treatment. As the bony partition between the cells and orbit is very thin, there is some tendency for the pus to enter the orbit and produce characteristic symptoms. Meningitis may result and death ensue. There may be suppurative ethmoiditis without retention or pressure-symptoms, the pus drying and forming crusts in the middle meatus, while periodical attacks of pain and swelling occur over the infraorbital region.

The **differential diagnosis** between empyema of the ethmoidal and other nasal accessory sinuses is by no means easy. A careful exploration with a probe should be made, hoping thereby to demonstrate the presence or absence of necrotic bone. This being done, the objective point in the treatment is determined.

Ethmoidal cells—empyema—treatment: When certain of the diagnosis, an opening should be made between the middle and inferior turbinate bodies with small sharp spoons. The direction of the opening should be outward, backward, and upward. After the ethmoidal cavities have been reached they should be explored with a probe for carious bone and other morbid material, which should be thoroughly removed by curettement. Irrigation with warm boric-acid solution should be practised in the after-treatment.

Sphenoidal Sinus—Empyema.

The **etiology** is obscure.

The **symptoms** vary with the degree and direction of pressure. There may be deep-seated pain, tinnitus, dizziness, exophthalmos, strabismus, deafness, and sudden blindness from pressure upon the optic nerve as it passes over the sphenoid through the optic foramen. Pus discharging from the postnares into the vault of the pharynx may be regarded as a sign of sphenoidal or posterior ethmoidal empyema.

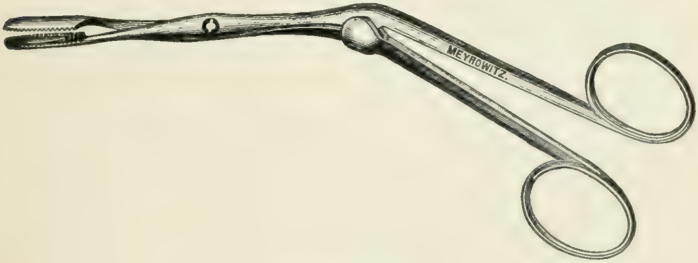
Sphenoidal sinus—empyema—treatment: The treatment is necessarily radical. Free drainage and irrigation of the sinuses are the objective points aimed at. My own experience has been limited to two cases, in both of which it was impossible to reach the sphenoidal foramen without first performing middle turbinotomy. After the removal of a large portion of the middle turbinal a Eustachian catheter was introduced into the sinus. The sinus was irrigated daily in this manner for several weeks until a cure was effected. After having gained entrance into the sinus it should be carefully explored with a suitable blunt probe for necrotic bone, neoplasms, etc. These, if found, should be removed by curettement, a specially devised spoon being required for the purpose. In some cases it may be necessary to enlarge the sinus-opening before treating the sinus-cavity. This may be done with one of the small Grünwald spoons.

FOREIGN BODIES IN THE NOSE.

Beans, corn, pease, beads, nails, and other small articles are sometimes put into the nose by children and insane people. In one case the author removed the nipple and percussion-cap-tube, weighing 325 grains, from the nose of a man sixty years old. It had been in his nose for thirty years.

The history developed the fact that thirty years ago, in firing a musket it exploded, destroying his left eye. Since that time he had been troubled with a foul-smelling pus-discharge from that side of his nose. It was for this condition he was referred to me. A pair of heavy angular forceps (Fig. 98) was used in its extraction. The smaller and more usual foreign bodies may be removed with a small hook (similar to an ear-hook), forceps, snare, or probe.

Fig. 98.



Angular forceps.

Rhinoliths: Should a foreign body become encrusted with carbonate and phosphate of lime it is called a rhinolith. Mucus, a blood-clot, a fragment of bone, etc., may become the nucleus of a rhinolith. The *treatment* is as for foreign bodies.

EPISTAXIS.

Etiology: Nasal hemorrhage may be due to traumatism, ulcers, and nasal tumors. In picking crusts from the septum or in forcibly blowing them from the nostrils the epithelial lining may be torn off and hemorrhage result. Epistaxis may also result from local congestion due to remote diseases, as chronic Bright's disease and affections of the spleen. Some of the exanthematous fevers (as smallpox, typhus, typhoid, and relapsing fever), purpura, scurvy, leucocythæmia, anosmia, plethora, acute yellow atrophy of the liver, and phosphorus-poisoning may be attended by nasal bleeding. In some cases it is due to a hemorrhagic diathesis.

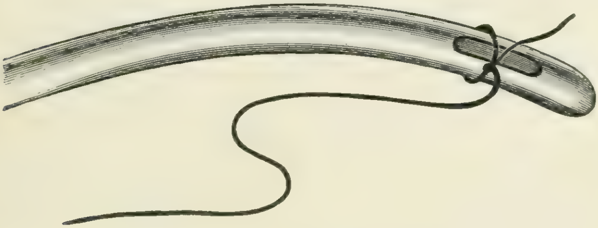
Epistaxis—treatment: In plethoric persons it may be well to allow the hemorrhage to continue for a time before attempting to check it. Mild astringents, as tannin, will check the hemorrhage in mild cases. Lying upon the back, raising the hand above the head, cold sponging to nose and forehead, or irrigation with water at about 110° F. are valuable remedies. The application of a saturated solution of antipyrine by means of cotton to the nasal membrane is often followed by speedy relief. In the more obstinate cases the nose should be carefully examined for bleeding points, which, if found, should be cauterized.

In cases which do not yield readily to the simpler methods of treatment, anterior and postnasal plugging should be done.

Anterior nasal plugging is best done with a long strip of gauze passed inward and upward between the posterior end of the middle turbinated body and the septum, where the end is held, preventing it from dropping into the pharynx. The remainder of the gauze should be packed in alternating up-and-down folds until the anterior nares is reached. This will usually check the hemorrhage, and should be left in place for from six to twenty-four hours.

Postnasal plugging is still more certain in its results. This is accomplished by passing a stout linen or silk thread *through the nose* into the pharynx by means of a Bellocq canula or a soft-rubber catheter (Fig. 99). The thread should be about three feet long, so that one end of it is sure to remain outside the nose. As the thread appears in the pharynx it should be seized with a pair of forceps and drawn forward through the mouth. It should then be cut loose from the canula or catheter, which should be withdrawn. A loop should be made in the oral end of the thread and a ball of cotton the size of an English walnut fastened within it. Traction upon the nasal end of the cord will draw the ball of cotton into the pharynx, the index-finger being inserted to guide it upward along the posterior pillar of the fauces into the nasopharynx. In this way the danger of inverting the soft palate into the postnares is avoided. Traction upon the cord, aided by the index-finger in the nasopharyngeal space, guides the cotton into the posterior nares, where by pressure it checks the hemorrhage. If necessary, strips of gauze can also be packed in the anterior nares. If the gauze is to be left *in situ* for more than twenty-four hours, it should be moistened with the compound tincture of benzoin, to prevent decomposition of the blood-clot and secretions.

FIG. 99.



Soft-rubber catheter and thread.

FUNCTIONAL DISORDERS.

Anosmia, or Loss of Smell.

Etiology: The loss of smell may be an intermittent or a permanent condition. In malaria, polypi, and rheumatic affections it may be intermittent. According to Sir Morell Mackenzie, if the anosmia lasts for two years it is likely to be permanent. The usual causes are inflammatory conditions of the nasal mucous membrane. Thus it is often present in hypertrophic and atrophic rhinitis. Nasal polypi may cause it by preventing the current of air reaching the olfactory area. It may be due to a lesion of the olfactory centre, or of the nerve itself. It may affect one or both sides.

The **treatment** of anosmia is successful in those cases due to syphilitic inflammation, malaria, hysteria, and temporary obstructions. It is unsuccessful in old-standing cases due to inflammation of the nasal mucous membrane and in cases of central origin. Insufflation of strychnine ($\frac{1}{24}$ gr. with sugar) has been recommended in obstinate cases, but in my experience it has proven to be useless.

Parosmia.

Symptoms: In this condition the patient complains of constant or recurrent perception of foul odors. It occurs in the insane, and is one of many other perversions of the senses observed among them. Persons affected by catarrhal inflammation of the mucous membrane of the nose sometimes complain of a foul-smelling and -tasting discharge which is odorless to others. It sometimes follows an attack of influenza.

The **treatment** consists of appropriate remedies and surgical measures addressed to the conditions causing it.

Hyperosmia.

Definition: This is a condition in which the sense of smell is more acute than normal.

It **occurs** in hysterical and very nervous persons.

The **treatment** should be addressed to the general underlying condition.

HAY-FEVER (Hyperæsthetic Rhinitis).

Synonyms: This condition is known under various names, viz., Vaso-motor Rhinitis, Vaso-motor Coryza, Summer Catarrh, Autumnal Catarrh, Rose Cold, Pollen-poisoning, etc.

It is **characterized** by annual recurrences of intense swelling of the nasal mucous membrane, attended by sneezing and profuse watery discharge from the nose. The eyes itch and burn, and are very sensitive to light.

Etiology: The chief *exciting cause* is the pollen of certain grasses. *Predisposing causes:* (*a*) a neurotic and rheumatic habit is perhaps the most important; (*b*) heredity, 35 per cent. having relatives similarly affected; (*c*) certain morbid conditions of the nose, as hypertrophic rhinitis, deflections and thickening of the septum, polypus and hyperæsthetic areas. There are, however, some cases in which there seems to be no local morbid condition. Some writers claim, however, that there are always three conditions which acting in unison cause hay-fever; that if one of these conditions is absent hay-fever does not develop. The three conditions are: 1. The irritating particles in the atmosphere. 2. The neurotic habit. 3. The local morbid condition of the nasal mucous membrane. If these three conditions are necessary to the existence of hay-fever, the removal of either one of them will effect a cure. In many cases the conditions are all present, and in some of them the removal of the local morbid condition effects a cure. It is, however, doubtful if it should be positively stated that the three conditions are present in all cases. While much has been written upon the subject, there is yet much to learn concerning the etiology and pathology of the disease. What we now know as "hay-fever" may in time be found to represent widely different conditions.

Hay-fever—symptoms: The *premonitory symptoms* are itching, smarting, and burning sensations at the inner canthus of the eye. Some complain of similar symptoms in the throat or roof of the mouth. The onset is usually sudden, so that within a few hours the case is well established. After the premonitory stage the inferior turbinate bodies swell with great rapidity, completely occluding the nasal passages. The eyes overflow with tears, the conjunctiva becomes injected with blood, and the nose pours forth quantities of serum and mucus. Various symptoms develop from time to time, as paroxysmal sneezing, photophobia, mental depression, spasmodic asthma, etc. The patient dreads the steadfast gaze of those about him. He is inclined to screen his eyes and shun the company of others.

The *objective* symptoms are those usually found in the various types of catarrh, septal deformities, and polypoid degeneration of the mucous membrane.

The **special pathology** of hay-fever is little understood. The pollen of certain grasses, flowers, etc., and the odor of certain animals excite the train of symptoms. In some cases there is hypertrophy of the inferior turbinate bodies, especially at their posterior extremities. Polypi are often present. In others, especially those suffering from asthma, there are numerous small elevated patches on the membrane covering the septum and inferior turbinate bodies. These are found less frequently in the meatuses and floor of the nose. The presence of uric acid and the xanthin group of toxins in the blood is regarded by some as the chief predisposing cause of the attacks.

Prognosis: This is favorable so far as life is concerned. The probability of permanent relief is good in some cases after the correction of the morbid conditions within the nose. Other cases will not improve under any form of treatment. If there is little or no local pathologic change within the nares, the prognosis under treatment is less favorable than in those cases in which there is marked intranasal disease. The prognosis should always be guarded, as the neurotic element is often quite prominent and renders the chance of permanent improvement quite problematical.

Hay-fever—Treatment.

General plan: Many remedies and methods of treatment have been advocated for the relief of this very distressing and persistent disease. None of them has proven to be of universal or even general application.

Those who favor the *neurotic origin* of the disease have advocated constitutional and diathetic remedies, with the hope that the nervous system when thus reinforced would no longer be affected by the phenomena presented in hay-fever.

Others have advocated the *pollen* origin of the disease, and have recommended a change of climate as the only effective remedy.

Still others hold that *local nasal disease* is the cause of hay-fever, and advocate the correction of all deformities, obstructions, and diseases of the nasal respiratory tract as the only rational method of treatment.

Clinical experience has demonstrated that in most cases all three factors must be combated in order to eradicate the affection. Oftentimes it is impossible to remove all, or even one, of the causes. In such cases a cure is impossible. In other cases the disease may be very much improved, or altogether cured, by either removing to a different climate, correcting the neurotic and lithæmic habits, or freeing the nose from morbid processes.

Hay-fever—palliative treatment: During the paroxysms or acute exacerbations curative treatment should not be attempted. The distress is so great, however, that palliative measures should be administered. Sneezing, lachrymation, asthma, and nasal stenosis are the most annoying symptoms. Remedies that will overcome the nasal stenosis are chiefly indicated, as the other symptoms are secondary to it.

Cocaine hydrochlorate in solution or powder varying in strength from 1 to 10 per cent. has been used with marked relief of all the distressing phenomena. There are *three objections* to its use, however, viz.: (*a*) After a time it looses its depleting effect and the engorgement becomes greater than before. (*b*) Its effects are transient and it must be used at short intervals. (*c*) The cocaine-habit is often quickly formed. Hence its use should be resorted to only after trying all other methods of treatment without success.

Various other remedies are used with more or less success. One will afford relief in some cases while it will be ineffective in others. Having tried one, another should be used, and so on through the list until one is found that gives satisfactory results. Sometimes a combination of two or more remedies is most effectual.

The author has used a warm solution of *boric acid* with a postnasal syringe (Fig. 39) in a considerable number of cases with success. The nostrils and nasopharynx should be thus washed every three hours until relief is afforded or its uselessness is demonstrated.

In some cases of hay-fever *Seiler's* or *Dobell's solution* may be used in the same manner, and with a like result. It is often of great advantage to add 1 per cent. of menthol to the solution.

Hydrozone (15-volume solution), or *pyrozone*, has been extolled by some writers as a valuable local application for the relief of these symptoms. My own experience with it has been limited to a few cases, and thus far has uniformly failed to give relief.

Menthol in oily solution or suspension applied to the nasal mucous membrane and to the postnasal space is a valuable remedy. It should be used in 2 to 10 per cent. solution. Sometimes it is essential to apply it to the posterior ends of the inferior and middle turbinals. This should be done with a curved cotton-wound applicator and the aid of the head- and the postrhinoscopic mirrors.

Scrubbing the nasal chambers with antiseptic solutions, as boric acid, Dobell's, or Seiler's solution has been recommended.

In simple hyperæsthesia attending the catarrhal type of rhinitis *massage* has proved of the greatest value in my hands. My experience with it in *hay-fever* has been too limited upon which to base an opinion. My practice is first to cleanse the nose with antiseptic sprays, douches, and the cotton mop; after which the membrane is massaged with a bit of cotton upon the end of a slender silver probe. The cotton is first dipped into some simple ointment to reduce the frictional irritation to the minimum. This method is theoretically correct, as it unloads the vessels and lymphatics and stimulates the vaso-motor and trophic nerves to a more normal activity.

Suprarenal extract promises great relief, as it rapidly and completely depletes the swollen turbinals. A saturated solution should be sprayed into the nose every three or four hours.

Linear scarification, as described under chronic rhinitis with turgescence, sometimes affords great relief. Cocaine anæsthesia should first be produced. Suprarenal extract should not be applied, as a moderate amount of hemorrhage is desirable.

Severe *asthmatic attacks* are often benefited by the administration of the following mixture (Kyle) every three or four hours:

℞. Morphinæ sulphatis,	gr. $\frac{1}{8}$;
Strychninæ sulphatis,	gr. $\frac{1}{60}$;
Hyoscinæ hydrobromatis,	gr. $\frac{1}{200}$.—M.

If the foregoing remedies fail to afford relief, the patient should be *sent to a suitable climate* for a period of two months, or until the "hay-fever" season is over. This does not effect a cure, but affords relief from the distressing symptoms. When it is impossible for the patient to leave home or business an inhaler made of sponge can be worn over the nose or within the nostrils, thus filtering the pollen and other irritants from the air. In this way complete relief is sometimes obtained.

Hay-fever—interparoxysmal treatment: During the intervals of freedom from acute manifestations the nose and general system should be brought to as near the normal as possible. Such treatment should not be postponed until the paroxysmal seizures appear. The nasal mucous membrane is then in no condition for surgical interference, and the general system will usually not respond promptly enough to abort the attack. As there are ten or eleven months of freedom from acute symptoms, this time should be used in preparing the patient to withstand the peculiar irritation that causes the paroxysms.

The *rheumatic* and *neurotic diatheses* are often associated in this affection and should be corrected as far as possible by appropriate treatment. The diet should be so regulated that the lithæmic habit is overcome. This can be best accomplished by the adoption of a more limited dietary. It is not so necessary to avoid certain articles of food as it is to avoid eating too great a quantity. By correcting the lithæmic diathesis the neurotic habit will also be relieved. Metabolism is better performed and the blood contains a smaller amount of toxic agents. The nervous system is therefore not subjected to so much irritation, and the patient becomes better able to withstand the local irritation from the inhalation of pollen, etc. The renal and intestinal excretions should be carefully regulated. One-half ounce of the granular effervescing sodium phosphate (Kyle) should be given every night in a glass of cold water. The kidneys should be stimulated by the administration of potassium acetate or some other simple diuretic.

One of the alkaline or lithia waters should be freely used to counteract the hyperacidity of the blood. Iron and arsenic should be given to tone the nervous system. The neck and chest should be douched with cold water every morning.

Intranasal irregularities and *hypersensitiveness* should also be corrected during the quiescent period. During the winter and spring months the mucous membrane of the nose is in the most favorable condition for surgical interference. Spurs, ridges, hypertrophies, hyperplasias, deflections, excrescences, polypi, etc., should be removed or corrected by appropriate surgical and remedial measures. These have been described elsewhere and will not be further considered here.

The *hypersensitive areas* should be located with a blunt probe and cauterized with a white-hot platinum knife. The areas may be located by passing the probe over the mucosa; the moment a sensitive area is touched the patient will experience intense burning, smarting, and itching sensations in the nose and inner canthus of the eye. A flat cautery-point, previously adjusted so that it will come to a white heat the moment the current is connected, should be applied. The flash of white heat will make a superficial burn and will be unattended by pain. If the electrode is heated to only a cherry-red, the tissues will be deeply burned and there will be intense pain. With the ordinary electrodes three storage-cells are required to produce instantaneous white heat. From two to four spots may be cauterized at a sitting. After a period of four to seven days the treatment may be repeated with safety. It should be continued until all sensitive spots are removed. The posterior ends of the turbinals and septum should be carefully searched for sensitive areas, and if found should be cauterized with the aid of the postrhinoscopic mirror.

Cocaine should not be used in the reduction of the sensitive areas, as it would interfere with the symptoms which enable the operator to locate them.

If the foregoing methods of treatment are judiciously applied, there is a fair prospect of cure in a considerable number of cases. It is well, however, to hold the patient under observation for about three years before assuring him of a permanent cure. In this way slight irregularities and morbid conditions can be corrected from time to time, and thus enable the operator to effect a cure in cases which would otherwise be attended by failure. The prognosis should always be guarded.

NEOPLASMS.

Myxomata (*Polypi*)*:* This subject has already been considered on pages 297 and 309.

Papillomata of the Nose.

Synonyms: Warty or Cauliflower-like Tumors.

Definition: These are benign growths which appear at the anterior margin of the nares, at the junction of the skin and mucous membrane, or upon the mucous membrane itself. They are usually unilateral.

Their **cause** is not definitely known. Excessive physiologic activity, especially when motile in character, as in the larynx, is sometimes given as a cause for their appearance. This sort of functional activity, however, does not exist in the nose.

Treatment: As the tumor is benign, it need not be removed unless it is the source of obstruction or irritation to the respiratory tract. If, however, their presence is a source of obstruction and irritation, they should be removed after thorough cocainization of the parts. A knife, the cold-wire snare, or cautery may be used for this purpose. As the growth is more or less pedunculated, the operation is easily performed. If the knife or snare is used, the wound should be cauterized to prevent recurrence of the growth.

Adenoma of the Nose.

This type of tumor in the nares is rare, as there is but little glandular structure in the mucosa of the nose. When simple it is benign, but it more often occurs in mixed tumors of the cancerous type. When it occurs as a simple adenoma it is composed of true glandular tissue, and not of lymphoid tissue, of which there is an abundance in the nasal mucosa.

Cystoma of the Nasal Passages.

Definition: This is a cystic formation of the nasal mucosa from degeneration of a lymphoid nodule; or from closure of the mouth of a mucous gland. When formed from a lymphoid nodule, it is the result of a degeneration by which the contents of the mass are liquefied, and a limiting membrane is formed from inflammatory reaction at the periphery. The appearance is like that of a polypus, except as to the fluid contents.

The **treatment** is that of polypus.

Angiomata of the Nasal Passages.

This condition is rare, which is rather remarkable in view of the fact that the nasal mucosa is very vascular.

Etiology: Angiomata are due to nutritive changes in the walls of the bloodvessels, rather than to changes in the mucosa. The lymphatic temperament is a predisposing cause.

Symptoms: Epistaxis occurs frequently. The presence of the tumor excites considerable irritation, as is shown by the mucopurulent discharge. The nose does not usually show external deformity. The tumors are red or purplish according to the predominance of arterial or venous blood within them. They are irregularly nodular, and often bleed profusely from slight wounds inflicted during examination.

Prognosis: Their development covers a period of from two to five years. Life is not usually greatly endangered, although hemorrhage may at times be alarming or even fatal.

Angiomata of the nose—treatment: Their removal is not followed by recurrence. The cold snare adjusted at the base of the tumor and very gradually tightened affords the safest means of operation. An hour or more should be used for this purpose, as haste might be attended by serious hemorrhage. The application of a freshly prepared solution of a dried extract of suprarenal capsule immediately after cocainization of the parts will contract the small arterioles and greatly diminish the amount of hemorrhage.

Chondroma of the Nasal Passages.

Occurrence: This form of tumor is rarely seen in the nasal passages. The few cases reported have occurred chiefly during the period of adolescence. The growth is composed of hyaline cartilage and may vary greatly in size.

Symptoms: Upon inspection it appears as a round, hard tumor, and very much resembles a fibroma. When of considerable size it gives rise to stenosis and mucopurulent discharge. Chondromata are usually located at the junction of the cartilaginous septum with the anterior nasal cartilages. They are benign and give rise to no serious symptoms beyond those due to nasal obstruction.

Treatment: They should be removed when thus offending.

Fibroma of the Nasal Passages.

This is a rare tumor composed of fibrous tissue.

The **etiology** is obscure. Inflammation and disturbed nutrition of the parts seem to favor development. About two-thirds of the cases occur in males. While the tumor may occur at any age, it is most often found between the ages of fifteen and thirty.

The **symptoms** are those peculiar to nasal obstruction. In appearance the growth is smooth, hard, and rounded in contour, and is covered with healthy mucous membrane. Epistaxis is a prominent symptom, and may be so severe as to render the prognosis grave. It invades any kind of tissue, and hence may grow in any direction.

Prognosis: The prognosis is grave unless the neoplasm is thoroughly removed. Its rapid, relentless growth through the adjacent tissues renders it a serious menace to life, as it may invade the cranial cavity and other important structures.

Fibroma—treatment: The treatment is surgical, the snare (cold or hot) perhaps affording the best means for its removal. It may be necessary to do the operation under a general anæsthetic, turning the lip and nose of the patient upward over the brows so as to expose more fully the nasal cavities. The danger attending the operation is from hemorrhage.

In other cases it may be necessary to make a temporary resection of the superior maxilla to expose fully the growth. This was done in one of the author's cases with good result, after first trying the cold-wire snare.

Osteoma of the Nasal Passages.

Definition and occurrence: This is a grave form of bony neoplasm which usually grows from the accessory cavities or the upper part of the nose. It involves other tissues lying in its path, and often causes marked facial deformity. Males are more frequently affected than females, and it is more common before middle life.

External deformity, nasal stenosis, and pain are the prominent **symptoms.**

Treatment: A radical external operation is usually required for its removal, after which it shows but slight tendency to recur.

Sarcoma and Carcinoma of the Nasal Passages.

Sarcoma: The rough surgical removal of nasal polypi is thought by some to be a *cause* of sarcoma. Catarrhal inflammation is a predisposing cause.

It usually *occurs* about middle life or at a much earlier period. Sarcoma of the nose runs a slower course, and is not so malignant as sarcoma in other parts of the body.

The prominent *symptoms* are nasal obstruction and repeated attacks of epistaxis.

Treatment in most cases consists in its removal by a radical external operation.

Carcinoma: This is a rare tumor in this locality, and as its treatment belongs to the general surgeon it will not be considered in this work.

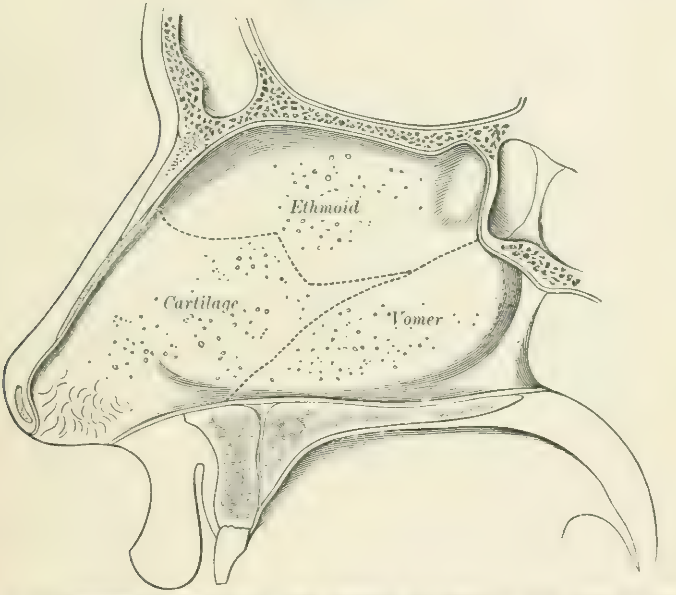
THE SEPTUM AND ALÆ.

Exostoses and Enchondroses.

Synonyms: Spurs; Ridges; and Crests.

Occurrence: These thickenings of the septum are composed of cartilaginous or bony tissue, or of both combined; and are usually located anteriorly on the lower third of the septum at the union of the cartilaginous and bony tissues. If the growth is in the form of a ridge or crest it usually extends from the anterior nares (where it is cartilaginous) backward to the bony portion of the septum (where it assumes the type of tissue from which it grows). On the bony portion (Fig. 100) of the septum isolated spurs of considerable size are often found, the presence of which cannot always be determined on account of a coexisting deflection of the septum or excessive hypertrophy of the turbinals. A spur growing in this location is often the source of considerable irritation and obstruction, and should be removed. Exostoses may also grow from the floor of the nostrils and the middle turbinal bodies. They

Fig. 100.



Anteroposterior section of the nose, showing the septum. The lines of junction of the several pieces are indicated by the dotted lines. The vestibule and the vibrissæ are also pictured. (Merkel.)

may be so large as to extend entirely across the nose to the opposite wall.

The **etiology** is not quite clear. In some cases exostoses appear to be of inflammatory origin, as chronic nasal catarrh or inflammation following traumatism. In other cases they seem to cause the catarrhal condition. In still other instances they are undoubtedly due to the dislocation of the parts composing the septum. Sometimes one side of the septum has a groove or depression corresponding to the ridge on the opposite side.

Spurs and ridges—symptoms: As they exert such an important influence upon the functions of the nose, and are so frequently present, the symptomatology will be given with considerable fulness.

When they are located upon the *anterior nares*, they obstruct the ingoing current of air. The diaphragm, acting upon the chest-cavity, causes the lungs to expand and air to rush in to fill the vacuum. The anterior nares being partially obstructed, the ingoing current of air does not gain entrance to the nares rapidly enough to prevent a partial vacuum being created. This leads to engorgement of the vascular structures of the mucous membrane and to overnutrition, as well as perverted nutrition, of the parts. The ultimate clinical picture is that of hypertrophic rhinitis and its attendant phenomena. The original source of obstruction is augmented by the increased vascularity and hypertrophy of the erectile tissue. The *respiratory* and *phonatory functions* of the nose are thus impaired. Catarrhal or even suppurative inflammation may result.

The mucous membrane of the *accessory sinuses* sometimes becomes the seat of inflammation and myxomatous degeneration.

The evil consequences of lost or impaired nasal respiratory functions are manifested by inflammatory and unstable functional activity of the lower air-passages. Postnasal catarrh, pharyngitis, laryngitis, tracheitis, bronchitis, and thickening of the endothelial lining of the air-vesicles may result. The latter is more fully explained under nasopharyngeal adenoids.

The *normal drainage* of the nose is impaired and the secretions are retained and decomposed, thereby forming a suitable soil for the growth of pathogenic organisms. Hence the inflammation often assumes a suppurative form.

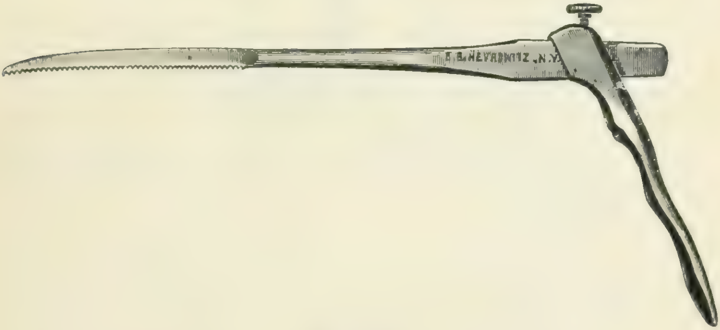
The *interchange of gases* which takes place through the nasal mucous membrane, as well as in the lungs, is interfered with on account of the obstruction and subsequent tissue-changes.

The *hearing* may be affected by thickening of the mucosa lining the Eustachian tube, thereby preventing normal aëration of the middle ear.

These phenomena, together with the usual signs of thickening of the cartilaginous and bony septum, form a *symptom-complex*, the cure of which is of vital importance to the well-being of the patient.

Spurs and ridges—treatment: In view of the disturbed functions of the respiratory tract, and secondarily of those of nutrition and tissue-metabolism, it becomes apparent that the radical removal of the spurs or other forms of thickening should be undertaken. Fortunately, the procedure in most instances is a simple one. The mucous membrane covering the spur or ridge should be anæsthetized by the application of a 4 per cent. solution of the hydrochlorate of cocaine. A thin film of cotton, large enough to cover the spur or ridge, should be placed upon the end of the finger and

FIG. 101.

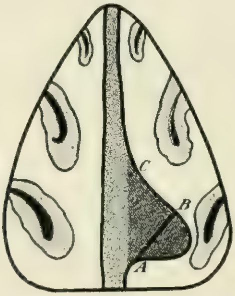


Mial's saw.

moistened with the solution of cocaine, and applied over the growth with a probe. It should be removed in from five to eight minutes. If the parts are not thoroughly anæsthetized, a second application should be made in a similar manner.

A Bosworth or Mial's saw (Fig. 101) should be introduced beneath the growth at its base, sawing from below upward, so as to leave the upper portion of the thickening unobscured by blood (Fig. 102). The mucous membrane forming the attachment above should then be severed with a bistoury or scissors. If the spur or ridge is quite large, the mucous membrane and periosteum should be incised and pushed aside, while the cartilaginous or bony tissue beneath is being removed.

FIG. 102.



Diagrammatic representation of a spur on the left side of the septum: *A*, point at which sawing should begin; *C*, point at which saw should emerge; *A B*, line along which the saw will travel *unless care be taken.* (Coakley.)

At the close of the operation, if the periosteum and mucosa have been pushed aside, they should be replaced and held in position for twenty-four hours with a gauze dressing moistened with the compound tincture of benzoin. Aristol or nosophen insufflated with the powder-blower, shown in Fig. 103, may be used as a subsequent dressing.

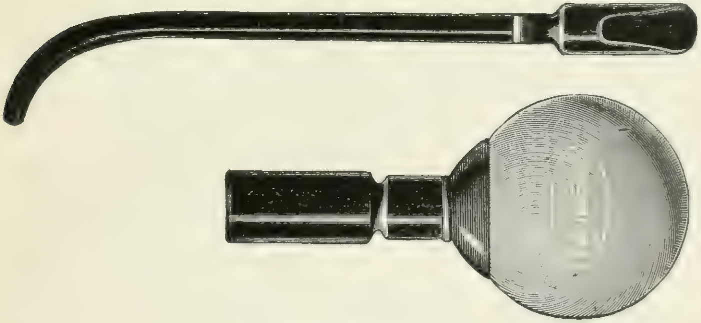
When the spur is located far back on the bony septum, it is often quite dense, and may require considerable time for its removal. Care should be exercised in the to-and-fro motion of the saw that other parts are not injured by the distal end.

Simple cartilaginous spurs of small dimensions may be removed with a curved bistoury. The wound remaining after these operations generally heals within a few days.

If the inferior turbinals are turgescent, a soft-rubber splint, devised by Richard Lake, or a thin sheet of celluloid or gutta-percha, should be introduced and worn until the healing process is complete, otherwise adhesions may form between the inferior turbinal and the freshly wounded surface.

Cartilaginous spurs can be removed by *electrolysis*, as first suggested by Minot. The bipolar method affords the best results. Needles connected with the negative pole should be thrust through the base of the spur, while those connected with the positive pole should be introduced at its apex. A current of from 15 to 25 milliampères should be applied for from fifteen to thirty minutes. The tissues are broken down by the electrolytic action of the current, while the process of disintegration is still further enhanced, as first pointed out by the author, by the liberation of certain chemical agents within the tissues. This secondary electrolytic action will continue for a considerable period after the primary electrolytic action has ceased. After from two to five days the spur will have become disintegrated and absorbed. In some cases it is necessary to repeat the procedure to effect a complete removal of the growth. The portion of the needle not introduced within the growth should be vulcanized or covered by small rubber

Fig. 103.



Powder-blower.

tubing to prevent painful contact with the skin at the entrance to the nose. The parts should be thoroughly cocainized as for other operations.

Deflection of the Nasal Septum.

Etiology: The etiology of this very common condition is still open to discussion. It is safe to conclude, however, that it is sometimes due to traumatism received during childhood or at a later period of life. It is rarely congenital, and is not often seen before the seventh or eighth year. As the septum is formed by the union of the vomer below and the perpendicular plate of the ethmoid above, deflection may result from excessive development of one or both of these bones. Deflection may also occur from pressure of the air-current during respiration. The middle or inferior turbinal upon one side being enlarged, the septum is pushed to the opposite side by the ingoing and outgoing currents of air. In other words, the deflection may be a compensatory effort to equalize the respiratory chambers of the nose.

Deflection of the nasal septum—symptoms: These are incident to nasal obstruction, and have been described elsewhere.

Upon *rhinoscopic examination*, the deflection may be seen as an angular or curved convexity upon one side and a corresponding angular or curved depression upon the other. It may be limited to the cartilaginous, although it usually involves the bony septum as well. It may be horizontal or perpendicular, or a combination of the two. Strings of ropy mucus or mucopus may be seen hanging from the septum to the inferior turbinal upon the obstructed side. This is a sign that the turbinal body has recently been in contact with the septum. Mouth-breathing, more or less complete, and the phenomena which attend it, are present. Upon the side of concavity there may be compensatory hypertrophy of the turbinal bodies. When this is the case a portion of the compensatory tissue should be removed before undertaking the correction of the deflection, otherwise the result of the operation will be unsatisfactory, as the septum will gradually return to its former position; or the side of concavity will be obstructed.

Treatment: The relief which follows correction of deflections of the septum is marked, and fully justifies the surgical procedure necessary for its accomplishment. Experience has demonstrated that only a few methods of operating are followed by permanent relief, the septum often returning to its former position. In view of this fact, it is important that a proper method of operating be chosen. This will be controlled largely by the degree, type, and location of the deflection.

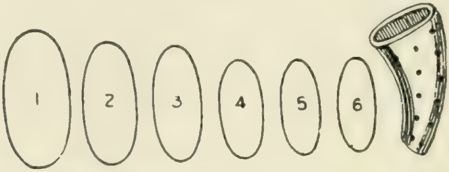
Operative measures should not be resorted to simply because the septum is deflected or thickened, but to overcome stenosis and to equalize the respiratory space within the two nostrils.

If there is a thin bulging deflection of the cartilaginous portion of the septum, it may be corrected by **gradually increasing pressure** applied for a period of three or four weeks. This may be done by means of Kyle's malleable nasal tubes, which can be gradually widened or narrowed as the case may require. They should be worn for several hours daily. Their presence excites inflammatory thickening of the septum and thereby strengthens it.

Where there is an angular ridge or deflection upon one side, the other being almost perpendicular, a **semilunar incision** of the mucosa should be made just beneath the ridge, thus forming a mucous flap, which should be dissected up and the ridge of cartilage beneath removed with a knife or saw. Great care should be exercised to avoid injury to the mucous membrane on the opposite side, as perforation might follow. The cartilaginous ridge may be dissected out with Asch's or with some other sharp-bladed knife which is curved on the flat.

The **Gleson operation** consists of a U-shaped incision. It is made so as to include the whole of the deflected or bulging portion of the septum except at the top, where the legs of the U-shaped incision end. The tongue, or flap, thus formed is pushed through the opening to the concave side, where it is held by impinging upon the sharp edge of the bevelled opening. The bulging or convex surface is thus carried toward the concave side and away from the obstructed side. Care should be taken to push the "tongue" completely through the opening. There will be little difficulty in pushing the lower portion of it through, but considerable force and care will be required to force the posterior and anterior borders through.

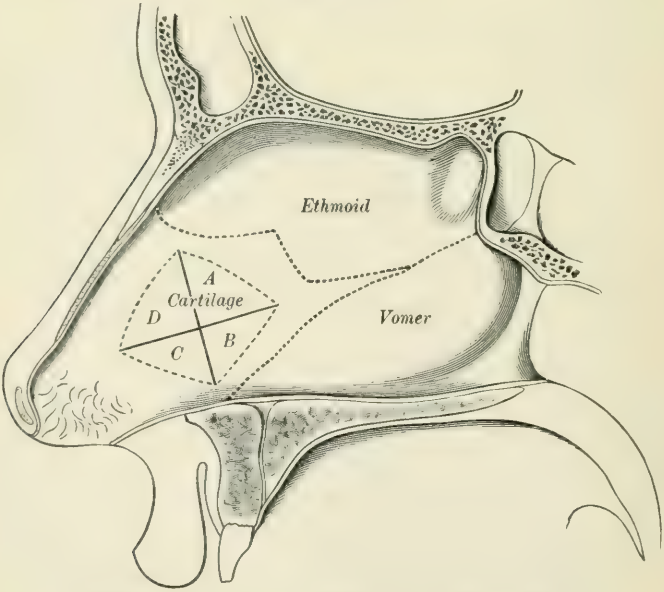
FIG. 104.



Mayer's hollow nasal splints. The cut shows one tube in profile and the exact diameters of the larger anterior ends of the six tubes of the set. The length of the smallest tube is one inch. The largest is one and one-half inches long. The others are intermediate in length.

The legs of the U-shaped incision should be made to extend as high up as possible, so as to loosen all or nearly all of the

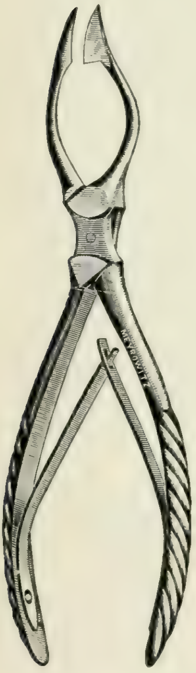
FIG. 105.



Septum showing the two incisions in the Asch operation.

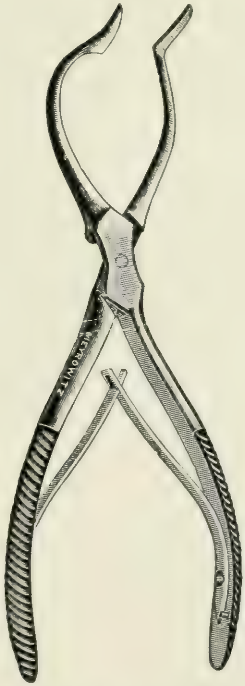
deflected portion. The neck of the flap should be fractured by pressure with the index-finger and the use of Adams' septum-forceps. The fracture will be attended by a sharp snap of the bone. A Mayer tube (Fig. 104) should be inserted and worn constantly for a week or more as a support to the newly placed tissues. It should be removed every two days and cleansed. After the first ten days it should be worn for one or two hours daily, and may be inserted by the patient.

Fig. 106.



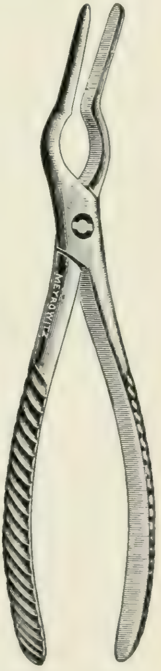
Asch's straight scissors.

Fig. 107.



Asch's angular scissors.

Fig. 108.



Asch's septum-forceps.

The **Asch operation** consists of a vertical and a horizontal incision (Fig. 105) through the septum, the incisions crossing near their centres. They are made with scissors having short blades somewhat like those used by tailors for cutting buttonholes. The scissors (Figs. 106–108) for making the horizontal incision are straight, while those for making the perpendicular incision are set at an angle to the shaft, thereby allowing them to assume a perpendicular position in the nose.

The patient should be placed under a general anæsthetic, and the parts painted with a freshly prepared solution of suprarenal extract, to prevent excessive hemorrhage. Without this precaution hemorrhage is very profuse, although it is not necessarily dangerous. The patient should be placed upon the operating-table in the Trendelenburg position, with the head hanging over the end of the table. In case of hemorrhage, aspiration-pneumonia will be thus avoided. The two incisions (Fig. 105) should now be made. The straight scissors should be introduced into the nose with the narrow blade on the side of convexity, and the incision made in as nearly a horizontal position as possible. The tip of the nose should be well elevated so as to bring the anterior end of the incision higher above the floor of the nose than it would otherwise be. The perpendicular incision is then made with the angular scissors, crossing the former incision near its centre. Four triangular flaps are thus created (Fig. 105, *A*, *B*, *C*, *D*), their apices meeting near the centre of the bulging septum. The index-finger is now introduced upon the convex side and the triangular flaps forcibly pushed to the concave side.

A large Asch tube is then introduced to hold the septum in its new position. Hemorrhage usually subsides at once upon its introduction. It should be removed at the end of two days, the parts carefully washed with warm boric-acid solution, and a tube of slightly smaller calibre introduced in its stead. This should be removed every two days to allow thorough cleansing of the nasal chambers. At the end of five or six weeks its use may be discontinued. If it is used for a shorter time, the septum is liable to return to its former position.

Secondary hemorrhage sometimes occurs, and if after a trial of ice-sprays, tannin, etc., it does not cease, the nose should be firmly packed with strips of gauze moistened with the compound tincture of benzoin. The compound tincture of benzoin prevents decomposition of the secretions in the nose, and makes it possible to leave the dressing intact for two or three days.

These methods, or some modification of them, will prove successful in the majority of cases.

Where there is an angular deflection of the septum with considerable thickening along the line or crest of the deflection the removal of the *thickened crest* with the **Bosworth saw** is often all that is required.

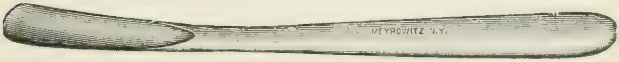
Sometimes **a spur** is present, and should be removed at a previous operation. It may be done at the time of the Asch operation, but is likely to be attended by perforation of the septum when thus performed.

Nasal Synechiæ (Adhesions).

Etiology: They may be congenital or acquired, and are composed of fibrous, cartilaginous, or bony tissue. They are usually caused by the contact and plastic union of two opposed inflamed surfaces. They may follow operations and cauterizations of the nasal mucous membrane, or they may be present in deflections of the septum.

Treatment by removal is required, as the band affords considerable obstruction to respiration. The band may be composed of bone-tissue and require the use of the saw for its removal. In other cases it may be mucous or fibrous tissue, and may be severed and separated with a gouge or elevator (Figs. 109, 110).

FIG. 109.



Asch's gouge.

FIG. 110.

Asch's elevator.

Some healthy tissue at either end of the synechia should be removed to separate the opposed surfaces as far as possible. Gauze or cotton dressing moistened with the compound tincture of benzoin or hydrozone should be frequently introduced to prevent contact of the surfaces. These dressings should be continued until the healing process is completed. In some of my cases the tendency to reunion was so strong that it was necessary to introduce vulcanized or soft-rubber splints between the surfaces. In two cases they were worn constantly for three or four months. They were, of course, removed every day or two and cleansed before re-introduction.

Collapse of the Nasal Alæ.

Etiology: It may be due to fault of formation of the lateral cartilages or to weakness of the dilator muscles of the nares, as in mouth-breathers. It also occurs in long, pointed noses with narrow openings, from contraction of the constrictor muscles of the nares.

Treatment: A perforated silver tube made to fit the anterior naris should be introduced and worn at least one-half of each day. Stenosis is usually greater at night, hence this is a favorable time for its introduction and use. It should be worn for several months, changing to a slightly larger size from time to time.

Ulceration of the Nasal Septum.

Etiology: Ulceration is due to local irritations from causes within the blood or from external influences. A lithæmic diathesis affords a local irritation, which, when the local predisposing conditions are present, lead to ulceration and perforation of the septum.

The *external influences* which cause it are the inhalation of dust-laden air, picking at the nose with the finger-nail, and the formation of desiccated crusts upon the anterior portion of the septum. A prominence upon the septum, as an exostosis or spur, predisposes to the formation of the crusts, the current of air drying the mucus more rapidly here on account of its exposed location. Foreign bodies and pressure from intranasal growths may cause it. Syphilitic ulceration and perforation have long been recognized as occurring in the nose. Ulceration or perforation of the septum in the very young should excite suspicion of congenital syphilis.

Ulceration of the septum—treatment: This should be stimulating in sluggish ulcers, characterized by grayish indurated bottoms, while the more inflamed and boggy red ulcers should receive sedative applications. Mildly stimulating applications of a 4 to 8 per cent. solution of the nitrate of silver, or a 20 per cent. solution of chromic acid are suitable lotions for the grayish indolent ulcers. If these and other stimulating applications fail, the base of the ulcer should be curetted, thus converting it into an acute inflammatory process. Inflamed boggy ulcers should be treated with an ointment containing 2 per cent. of carbolic acid; 1 per cent. of menthol; and 3 per cent. of boric acid. The nostrils should be douched twice daily with Dobel's or Seiler's solution to prevent irritation from decomposed secretions.

Perforation of the Nasal Septum.

Etiology: Perforation occurs in about 0.5 of all cases under treatment for nasal disorder. Congenital perforation is very rare. The causes have already been given under ulcerations, of which perforation is but a later stage. Ulceration and perforation are due to (*a*) arrested or faulty development; (*b*) to local inflammatory action; (*c*) to traumatism; and (*d*) to some systemic poison, as syphilis, the infectious fevers, or the lithæmic diathesis. It is most common during middle life.

Perforation of the septum—treatment: Little can be done to close the opening. Crusts and ulcerated edges may form on the margins of the perforation and occasion considerable annoyance to the patient. Slight hemorrhages occur upon forcibly blowing the nose, or in attempting to remove the crusts with the finger-nail. These conditions can be rendered more tolerable by suitable treatment. To remove the crusts, have the patient use an alkaline solution with a nasal douche until they are softened and can be removed without disturbing the epithelium beneath. In this way hemorrhage will be avoided. Instillations of a 25 per cent. solution of hydrozone or the peroxide of hydrogen will also soften the crusts. A 3 per cent. solution of the nitrate of silver has served me well in the cure of the ulcers or abrasions on the margins of the perforation. Granulations should be touched with fused chromic acid crystals or the actual cautery.

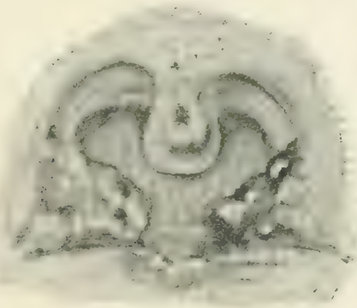
THE PHARYNX.

INFLAMMATIONS OF THE PHARYNX.

Acute Pharyngitis.

Etiology: Five per cent. of all diseases of the pharnx are acute inflammations. It is most common among the young on account of the strumous diathesis which manifests itself at that period of life. Either insufficient or an over-abundance of clothing should be taken into account in studying the etiology of the disease. An over-rich and abundant diet with hot drinks predisposes to it. Nasal and pharyngeal obstructions are, however, the chief causes of the malady.

FIG. 111.



Acute pharyngitis and tonsillitis with œdema of the uvula.

Acute pharyngitis—symptoms: The functional symptoms are painful deglutition, hawking or hemming, obstructed nasal respiration, temporary defective hearing, anosmia, and loss of taste. The *objective symptoms* (Fig. 111) embrace a dry, red, swollen membrane, which later is covered with a thick, tenacious mucus. This at a still later period becomes mucopurulent. The *constitutional symptoms* begin with fever (usually high in children), a dry coated tongue, constipation, indigestion, headache, and muscular and joint-pains over the whole body.

Acute pharyngitis—treatment: This should begin with the administration of calomel, followed by a brisk saline purgative. Aconite in one-drop doses should be given until the pulse goes down and the skin becomes moist. Antirheumatic remedies, as the salicylate of sodium, should be given to counteract the gouty diathesis. Vegetable tonics are indicated in some cases on account of the stomach disorder.

The *local treatment* should aim at the contraction of the blood-vessels. Lozenges of guaiacum, 2 grains each, are especially useful where there is a rheumatic diathesis. Menthol, $\frac{1}{4}$ grain, or menthol combined with oleum eucalyptol., forms a grateful and efficient paste or troche. Tannate of glycerin, steam inhalations, and applications of lunar caustic are of doubtful value. The sucking of ice or cold sprays are very grateful. When pain and great congestion are present equal parts of guaiacol and sweet oil, as a local application, are a most efficient remedy. A saturated solution of the suprarenal extract is also a very efficient local remedy in the very acute cases. It produces a smart burning sensation for a few seconds, but quickly relieves the congestion. Menthol, 20 parts, to 80 parts of olive oil, should be used in less severe cases. Externally wet compresses are sometimes of value. Scarification may become necessary when œdema develops.

Phlegmonous Pharyngitis.

Etiology: This type of pharyngitis develops when the patient is placed under unhygienic surroundings, as foul gases of the dissecting-room, the nursing of patients affected by erysipelas, foul wounds, and the exanthematous fevers. Drinking unsanitary water may cause it. Patients suffering from the exanthems occasionally develop this type of pharyngitis.

Phlegmonous pharyngitis—symptoms: The attack is usually ushered in by a feeling of languor, quickly followed by rigors, high temperature, a rapid pulse, and delirium.

The characteristic symptom is the suppurative inflammation involving the deep structures of the pharynx. The pus burrows beneath the deep cervical fascia or into the œsophagus. Œdema of the larynx may develop, and be attended by the danger of suffocation. Hemorrhage may occur on account of the sloughing.

The tonsils are often involved and may cause the disease to be mistaken for peritonsillar abscess. There is pain upon swallowing. Gangrenous sloughing may occur. The cervical glands are swollen. Streptococcus infection is usually found, although it is often a mixed infection.

The **prognosis** is unfavorable for the following reasons:

1. The patient may suffocate on account of œdema of the larynx;
2. The pus may burst into the œsophagus or the trachea;
3. Erosion of large vessels may cause fatal hemorrhage;
4. A fatal degree of septicæmia may develop.

The duration of the disease is from three to fifteen days. Paralysis of the palatine, faucial, and pharyngeal muscles may be a temporary sequel.

Phlegmonous pharyngitis—treatment: On account of the profound constitutional disturbance, general tonics, as iron, strychnine, quinine, etc., should be given. If great depression exists, stimulants in large quantities are indicated.

Local remedies: Wet compresses, ice sucked and swallowed, cold demulcent drinks, and lozenges composed of guaiacol (gr. 2), hydrochlorate of cocaine (gr. $\frac{1}{4}$), and menthol, are all valuable remedies in relieving the distress from tumefaction, and altered functions of the parts. Scarifications are of doubtful value, as the disease is of a serious infectious type. If it were a simple hyperæmia of the membrane, scarification would be indicated.

Tracheotomy should be performed if severe dyspnœa develops from extension of the œdema into the larynx. The toxæmia attending the disease is so great that even this radical measure does not always save the life of the patient.

Retropharyngeal Abscess.

Etiology: Lenox Browne reports that in thirty years' experience in out-patient work he has seen twice as many cases in adults as in children. Other writers say it is chiefly a disease of childhood. Caries of the cervical vertebræ has long been given as one of the chief causes, but a larger experience shows this to be untrue. The loose connective tissue beneath the pharynx is a favorable site for abscess-formations during the acute infective inflammations of the throat. Traumatisms, as from fish-bones and other foreign bodies, may also give rise to it. Occasionally it may arise from nasal or middle-ear inflammation. The syphilitic and strumous dyscrasiæ predispose to it.

Retropharyngeal abscess—symptoms: The subjective or functional symptoms may be classified as follows:

(*a*) The act of swallowing is interfered with;

(*b*) If the abscess is low down, dyspnœa becomes an important symptom.

(*c*) The respiration is stertorous; and if the larynx is involved it is stridulous;

(*d*) If the vertebræ are diseased rotary movements of the head cause pain;

(*e*) The temperature may or may not be elevated.

Objectively a fluctuating swelling may be seen upon the posterior wall of the pharynx. Aneurisms should be excluded before incision is made to evacuate the pus.

Prognosis: Those cases associated with caries of the spine are grave. In children there is in all types of pharyngeal abscess the danger of strangulation from rupture during sleep.

Retropharyngeal abscess—treatment: As soon as a diagnosis has been made the abscess-cavity should be freely lanced, the head thrown forward, and the pus expectorated. If the abscess is low down, it may be necessary to enlarge the opening with the index-finger. Local anæsthesia should be secured by applying cocaine. The incision should be made in the median line, so as to avoid injuring the internal carotid. If caries of the vertebræ is present the head should be fixed by a jury-mast, or other orthopedic appliance, to prevent frictional erosion of the diseased bones.

Chronic Pharyngitis.

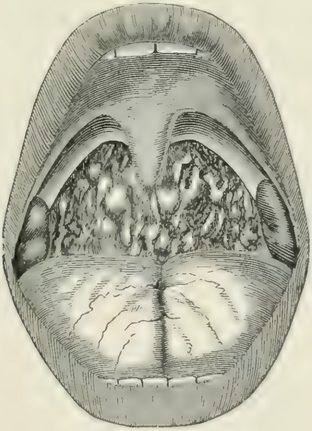
Synonyms: Clergyman's Sore Throat; Granular Pharyngitis.

Varieties and etiology: Chronic pharyngitis may assume one of four types, viz.:

1. *Diffuse inflammation* of the mucosa. This type sometimes follows the acute form.

2. Hypertrophy of the lymphoid tissue surrounding the pharyngeal follicles, or *follicular pharyngitis* (Fig. 112). As the lymphatic structures are more numerous on the lateral walls of the pharynx the nodular thickening is most prominent in these locations. The condition is sometimes referred to as *pharyngitis lateralis hypertrophica*. Mouth-breathing, struma, and other dyscrasiæ are factors in the production of this type of chronic pharyngitis.

FIG. 112.



Follicular pharyngitis.

3. *Exudative pharyngitis* is a modification of the follicular or granular type. It is usually thought to be associated with

the rheumatic diathesis. The secretion instead of being clear becomes opaque and of a pasty consistence, as in follicular tonsillitis.

4. The so-called *pharyngitis sicca* is not a true inflammation but a dry, glazed condition of the mucous membrane due to diseased conditions of the nose, as atrophic rhinitis or rhinitis with collapse of the erectile tissue as found in anæmic women; and to nasal stenosis. Perhaps a more appropriate name for the condition would be pharyngea sicca. Improper use of the voice and methods of breathing are also fruitful sources of chronic pharyngitis.

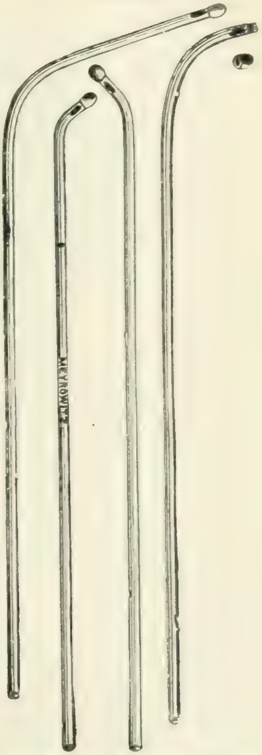
Chronic pharyngitis—symptoms: The patient usually complains of a sense of fulness in the throat, with slight pain or discomfort, and is troubled with hawking and expectorating on rising of mornings. When the second stage (simple follicular or granular) is reached the voice tires more easily, and the throat becomes more painful after slight use. The voice after a time becomes husky, necessitating frequent "hemming" to keep it clear. Cough is a troublesome symptom in all varieties, but more especially in the follicular and exudative types. The hearing is sometimes impaired by plugs of mucus in the Eustachian tubes, and more rarely by extension of the inflammation into the middle ear.

Chronic pharyngitis—treatment: It is well to *begin* the treatment by the administration of a saline cathartic, followed with iron, tonic doses of quinine, nux vomica, phosphorus, etc. The use of spirits, tobacco, and an over-rich diet should be interdicted. If the *uvula* is elongated, it should be amputated at about its middle portion. The local use of *vegetable astringents*, as rhatany, krameria, glycerite of tannin, etc., is of value in the simpler cases.

In the *follicular variety* the mineral astringents, as the perchloride of iron, sulphate of copper, nitrate of silver, and the ethereal tincture of iodine are of greater value than the vegetable astringents. The truth is that nothing short of caustic applications (Fig. 113) to the enlarged lymphoid nodules is of lasting benefit. London paste, lunar caustic, chromic acid, etc., have given way to the more convenient and thorough *electric cautery*. The small platinum wire point can be limited to a much smaller area than the mineral caustics. Before using either the chemical or electric cautery the pharynx should be cocainized.

In the *exudative type* the opaque secretion, with a small sharp spoon, should be squeezed or scraped out of the follicles, which should then be lightly cauterized with the fine platinum electrode brought to a red heat. Gouty and rheumatic remedies are indicated, more especially if the patient is of full habit, and has migratory pains in remote parts of the body.

FIG. 113.



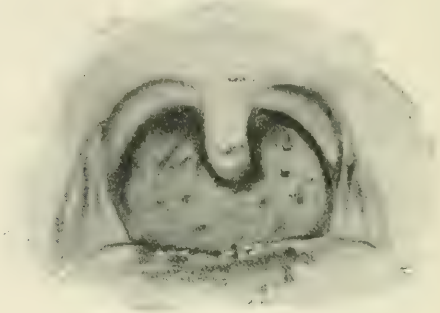
Aluminum applicators for applying caustics.

Pharyngea sicca, caused by general anæmia associated with collapse of the erectile tissue of the nose, should suggest the administration of appropriate remedies for the anæmia. If it is due to stenosis of the nose, nasal surgery should be resorted to for its relief. Local applications of a mildly aromatic and stimulating nature are grateful to the patient. They are best applied with an atomizer. Gargles are of little or on value unless it is in the diffuse variety, which may be regarded as subacute in character.

Tuberculosis of the Pharynx.

Pathology: This is a rare disease, and is usually secondary to pulmonary tuberculosis. A proper diagnosis is not always made, it being regarded as an acute catarrhal pharyngitis (Fig. 114). At first the membrane is red and infiltrated, and at a later period, yellowish spots or miliary tubercles appear upon its surface. After a time they break down, the secretion becoming mucopurulent. The posterior pillars of the fauces become stiffened from infiltration, thereby permitting the entrance of liquid foods into the postnasal space. Scar-tissue does not usually form in the wake of the ulcerative process, as only the superficial layers of the membrane are involved.

FIG. 114.



Fauces and pharyngeal wall in phthisis.

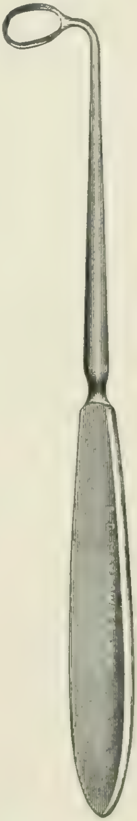
Symptoms: Pain is a constant symptom that gradually increases from a dull ache to sharp lancinating pains which are aggravated upon deglutition. The voice becomes thick and muffled. Cough is not more prominent than that due to the attendant pulmonary tuberculosis. The breath is fetid. The constitutional and local symptoms of pulmonary tuberculosis are present. Tubercular inflammation of the middle ear is often an extension of the process by way of the Eustachian tubes.

Prognosis: This is quite grave, death often following within a few months. If the process in the pharynx is primary, the prognosis is much more favorable. Primary tuberculosis of the pharynx is, however, an extremely rare occurrence.

Tuberculosis of the pharynx—treatment: There is little prospect of cure, except in the very rare primary cases. A 4 per cent. solution of cocaine painted over the inflamed surface gives temporary relief from the pain. Kyle reports that dilute nitric acid in equal parts of water is as effective as cocaine, and that the juice of pineapple sprayed into the throat affords great comfort. The thick, tenacious mucus and mucopus should be frequently removed from the pharyngeal wall by alkaline and antiseptic sprays and gargles.

The use of the curette (Fig. 115) on the infiltrated areas, if thorough and complete, may check the process. Residence in a suitable climate, and the internal administration of the carbonate of guaiacol or creosote are of positive value in the early stages of the disease.

Fig. 115.



Hartman's lateral curette.

Syphilis of the Pharynx.

About $3\frac{1}{2}$ per cent. of all diseases of the fauces and pharynx are syphilitic.

Forms: It may manifest itself in four forms—*i. e.* (*a*) primary, (*b*) secondary, (*c*) tertiary, and (*d*) congenital or hereditary.

The **primary form** is rare, as the fauces and pharynx are but slightly exposed, on account of their location, to primary inoculation. Chancre may be mistaken for tubercle and epithelioma, in both of which there is swelling of the lymphatic glands. It is usually recognized as syphilis only after constitutional symptoms develop.

The Secondary Form.

This **occurs** about one year after the primary infection.

Upon **inspection** a superficial ulceration or erosion of the membrane may be seen.

Etiology: Kaposi believes the condition to be still capable of infecting other individuals, and that many children feeding from a wet-nurse thus contract the disease from the secondary manifestation on the mammæ. Such cases are usually regarded as inherited syphilis.

The **pathology** of this stage is as follows: (*a*) erythematous patches, superficial infiltration, exudative plaques, or condylomata on the pillars of the fauces, uvula, and velum.

Secondary symptoms: Subjectively they are those of an ordinary sore throat. A striking peculiarity of this stage is the symmetrical appearance of mucous patches and erythematous areas on the two sides. The history of the case and the peculiar skin eruptions will generally enable the physician to make a correct diagnosis.

Tertiary Syphilis of the Pharynx.

Pathology: This form appears at about the third to the fifth year after the primary infection, as a gummatous infiltration which rapidly breaks down, forming an ugly ragged ulcer. The pillars of the fauces are most often affected, the loose tissue affording a favorable field for round-cell infiltration. Another site often affected is in the median line, at the junction of the hard and soft palate. The ulcers may appear upon the tonsils, tongue, roof of the mouth, posterior wall of the pharynx or buccal surfaces. The deep variety of gumma is more circumscribed, and when it breaks down is more destructive. This form is responsible for most of the cicatricial bands remaining in the wake of syphilis of the fauces and pharynx.

Congenital Syphilis of the Pharynx.

Occurrence: About 50 per cent. of the cases become manifest within the first five years of life. It occasionally appears in adult life.

Gross pathology: An ulcer appears with indurated edges. Its favorite site is the palate, although it may appear in any other situation. If the palate is the part involved the nares may be invaded by direct extension through the floor of the nose. According to Lenox Browne: "The fauces, the nasopharynx, the posterior pharyngeal wall, the nasal fossæ, the septum nasi, the tongue, and finally the gums" are affected in order of frequency. In the secondary form the diseased areas tend to affect both sides symmetrically, while in this form the ulcer is almost always in the median line. It also has a predilection for the bony structures.

Influence of congenital syphilis upon infectious diseases of childhood: Lenox Browne sums up the evidence on this point accumulated by syphilographers as follows: "(1) That while congenital syphilis affords no absolute protection against certain acute infectious diseases, its existence in the individual seems often to mitigate their severity; and (2) that certain acute diseases, accompanied by an exanthem—as scarlatina and measles—favor the dissipation, at least temporarily, of the pharyngeal and other manifestations of syphilis. On the other hand, when diphtheria supervenes during the existence of syphilitic lesions in the throat, the patient is liable rapidly to succumb."

Congenital syphilis—symptoms: They vary according to the extent and locality of the ulceration or necrosis. If the roof of the mouth is perforated, or if the pharyngeal muscles which shut off the pharynx from the nasopharynx are affected, the child cannot nurse or take the bottle. Coryza, excoriations, and scabs about the nostrils and upper lip may also be present. The child has what is popularly called the "snuffles"; others call it "the decay," on account of the malnutrition which is so noticeable in these sufferers.

Syphilis of the Pharynx—Treatment.

The **secondary stage** calls for the administration of mercury in mild doses, with especial care to avoid producing ptyalism. Direct the patient to wash the mouth thoroughly with a solution of the chlorate of potassium. Local applications of caustics, as acid nitrate of mercury, are of value in the active ulcerative stage. Iodine vapors or nebulæ exert a beneficial influence in those cases extending to the Eustachian tube and middle ear. Insufflation of iodol powder on the ulcerated spots has superseded the use of iodoform, as its odor is less offensive. Amputation of enlarged tonsils should not be done during this stage, as the raw surface thus created would very likely assume the syphilitic form of ulceration.

The **tertiary stage** calls for the so-called mixed treatment. The iodide of potassium should be administered during the ulcerative stage.

It is wise to begin with about 3-grain doses and gradually increase them to the point of toleration. After the subsidence of the acute manifestations some form of mercury should be administered for about two years in order to prevent a recurrence of the destructive process.

Local applications of mineral caustics to the deep ulcers is to be recommended. The galvano-cautery is, however, a better method. Gargles of the permanganate of potash, the chlorate of potash, etc., exert a favorable influence upon the morbid process, and keep the surfaces free of detritus, which would, if left *in situ*, excite irritation.

The **congenital form** should be treated about the same as the acquired. As children tolerate larger doses of mercury proportionately than adults, this remedy may be pushed more vigorously. The nose, mouth, and throat should be kept free from crusts by douches and sprays, after which the throat should be sprayed with oily solutions containing menthol, camphor, and eucalyptus.

A Comparison of Syphilitic and Tuberculous Ulcers (Lenox Browne).

Syphilitic Ulcers.	*Tuberculous Ulcers.*
Deeply excavated.	No apparent excavation.
Few granulations, highly inflammatory.	Much indolent granulation.
Deep-red areola.	Faint areola.
Sharply cut edges.	Irregular and ill-defined edges.
Distinct demarcation.	Demarcation indistinct.
Yellow purulent secretion.	Grayish, ropy mucous secretion.
Discharge profuse.	Discharge scanty.
Penetrating to deeper tissues.	Superficial, with lateral in place of deep extension.
No fever.	High fever.

Nasopharyngeal Catarrh, or Chronic Postnasal Catarrh.

Etiology: This is a chronic inflammatory condition of the vault of the pharynx which is usually associated with, and is probably secondary to, some form of intranasal disease. It is also associated with certain other morbid states of the nasopharynx. According to Bosworth there is no physiologic connection between this form of catarrh and catarrh of the lower pharynx, as the pharynx is a part of the digestive tract rather than a part of the respiratory tract. Nevertheless it seems that the irritation resulting from the discharges from the nasopharynx must to a certain extent cause the catarrhal process in the oropharynx. Catarrh of the nasopharynx is rarely a primary affection. It is almost always secondary to intranasal affections and adenoid disease of the nasopharynx. The following are the *diseases* usually responsible for the condition :

1. *Nasal stenosis* from hypertrophy of the turbinated bodies, more especially of their posterior extremities.
2. *Polypi* causing stenosis and suppurative rhinitis.
3. *Empyema* of the *posterior ethmoidal cells* discharging posteriorly.
4. *Empyema* of the *sphenoidal sinuses* discharging posteriorly.
5. *Dry rhinitis.*
6. *Atrophic rhinitis.*
7. *Simple chronic rhinitis* with collapse of the erectile tissue, as found in anæmic women and children.

The *miscellaneous causes* of postnasal catarrh are as follows :

1. Pus discharged through the Eustachian tubes during otitis media suppurativa.
2. Follicular or other disease of the postnasal lymphoid tissue.
3. Disease of the pharyngeal bursa.
4. The lymphatic temperament or diathesis.
5. Excessive use of alcoholic stimulants.
6. The weather, season, and clothing.

Nasopharyngeal catarrh—symptoms : They will depend upon which of the foregoing causes is operative in its production. In addition to the nasal, ear, and other symptoms, the patient complains of a discharge (pus and mucopus) from the upper pharynx. This is usually described as "a dropping." If the pus is from the nose it has a thick yellow quality ; while if it is from the pharyngeal bursa it is more fluid. It may accumulate and dry during the night, causing morning exacerbations of nausea and vomiting in the endeavor to remove it.

The **diagnosis** should be addressed more to differentiation of the causes than to the local condition *per se.* The local condition is to be regarded as a symptom of one or more of many causes.

The **prognosis** depends upon the ability of the examining physician to properly locate the cause and relieve the same. If the intranasal or other causes are removed the cure of the postnasal catarrh is reasonably certain.

Nasopharyngeal catarrh—treatment: In view of the foregoing statements it is apparent that local washes, sprays, and astringent applications afford but temporary relief in the great majority of cases. *The treatment must be addressed to the removal of the intranasal and other causes.* The treatment of the nasal accessory sinus-, ear-, and adenoid-diseases are considered elsewhere, and will not be further elaborated here.

Local alkaline and antiseptic washes used with the postnasal syringe afford marked relief. Seiler's or Dobel's solutions are easily obtained and are well suited for this purpose. Astringent applications to the nasopharynx of glycerotannin, argentic nitrate gr. x to xxx to the ounce are regarded by some as efficient remedies. The following mixture, sometimes spoken of as the Vienna mixture, has proven a valuable addition to the local treatment in the practice of the author:

℞. Iodin. cryst.,	gr. x;
Potass. iodid.,	gr. xxx;
Glycerin.,	℥j.—M.

Sig. To be applied to the nasopharynx with a cotton-wound applicator after cleansing with Seiler's or Dobel's solution.

If alcohol is used to excess, it should be interdicted. The clothing should receive proper attention, and morning plunge baths recommended for their tonic effect.

The thickening in the vault of the pharynx should be treated according to methods given under *Adenoids.*

DIPHTHERIA.

Definition: It is an acute infection attended by constitutional and local symptoms, due to a specific microörganism, known as the Klebs–Löffler bacillus. The infection is rarely simple, but is usually "mixed" with streptococci, staphylococci, diplococci, and tetragoni. It is very generally held that the disease is more virulent when there is mixed infection, and more especially if streptococci are present in large numbers.

The Klebs-Loffler bacillus: This is a rod-shaped bacillus varying in length from 1.5 to 6 micromillimetres, and in width from 0.5 to 0.8 micromillimetres. These bacilli are usually slightly curved and somewhat club-shaped at the ends. They are usually arranged in pairs, either parallel or at an angle to each other, forming figures somewhat like the letters V, N, M, W, or X. They are sometimes single, and rarely end to end, forming long threads. They stain readily by Gram's method and Löffler's methylene-blue.

Bacilli having the same physical characteristics as the Klebs-Löffler bacilli, but without power to cause the phenomena known as diphtheria, are called non-virulent Klebs–Löffler bacilli. They may become virulent under certain conditions not yet well understood.

Diphtheria—etiology: 1. The cause is the *Klebs-Löffler bacillus.* It was formerly thought that these bacilli remained localized in the fauces or other part of the body primarily affected; but they have been found in the blood, glands, intestines, and other organs of the body. They are, however, rarely found in any considerable number except at the point of initial infection. They find in the fauces, nose, etc., a suitable soil and temperature for their growth, and are propagated in great numbers. The toxins formed in consequence of the presence of the bacilli are absorbed and carried through the lymphatics and bloodvessels to every part of the system. In this way the various general symptoms are developed. The fever and great depression are due to the action of the toxins upon the nerve-centres presiding over the production of heat and the processes of metabolism.

2. *Age* has an influence upon the susceptibility of the individual to the disease. While it may occur at any age, 95 per cent. of the cases are in persons under ten years; and most of these occur between the first and fifth years. Those occurring in adults, as nurses, mothers, and others, are almost invariably affected with either nasal stenosis or enlarged tonsils.

3. *A cold, moist climate* is more favorable for its development than the warmer, or the extremely cold ones. The spring and fall of the northern temperate latitudes on this account are especially visited by this disease.

4. Certain *predisposing* and *systemic conditions* in a measure

account for the prevalence of diphtheria in the cold, damp temperate climates. For instance, catarrhal diseases, lymphoid hypertrophy in the fauces and nasopharyngeal space, are more prevalent in this sort of climate, and predispose to the diphtheritic infection.

5. *Sanitation* should receive more consideration as an etiologic factor in diphtheria. Soil that is badly drained, in which there are decaying vegetable and animal matter, affords a nidus from which it may spread and become endemic. Plenty of *pure fresh air* is a foe to the disease, while the stagnant, dead air so often found in schoolrooms and sleeping apartments adds to its virulence and tendency to spread. *Sunshine* is rarely mentioned in this connection, but its absence from the schoolroom, home, and other environment of children may cause widespread dissemination of the disease. In one of the great London Children's Hospitals a few years ago there were certain wards in which all the diphtheritic patients were kept until all signs of the disease had been absent for a few days, when they were transferred to other wards. No recurrences among these transferred cases were reported until the outer heavy steel blinds were closed on account of repairs on an adjoining building. A few days after the sunlight was thus shut out, recurrences became the rule rather than the exception.

The contagion may be *transmitted* by sewer-gas, privy vaults, railway trains, winds, drinking water, milk, domestic animals and fowls, and from one person to another.

Diphtheria—pathology: The bacillus grows upon the mucous membrane and the toxins are elaborated and absorbed into the general circulation. The toxins are regarded by some as enzymes liberated by the bacilli. They digest the proteids of the body, thereby forming toxic albumoses. "The diphtheria toxins do not all originate in the membrane, nor do the albumoses merely accumulate in the tissues; but probably the digestion of the body-proteids, by the action of the enzymes absorbed from the membrane, forms the toxins. These poisonous substances are of two kinds, namely, albumoses and an organic acid, which are responsible, by their action on the nervous system, for all the symptoms—the fever, the cardiorespiratory asthenia, albuminuria, and paralysis."[1]

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[1] L. Browne, 5th edition, p. 499.

The first effect of the toxin is to produce a local œdema and irregular temperature.

The *false membrane* is composed of three layers, namely: (*a*) the outer or superficial layer, which contains a network of fibrinous exudate, numerous bacilli, and a few leucocytes; (*b*) the middle layer, which contains a fibrinous exudate, many leucocytes, and few bacilli; (*c*) the inner layer, which is modified mucous membrane, the coagulated fibrin having penetrated the mucosa and replaced the surface epithelium.

Round-cell infiltration occurs in the mucous and submucous tissue. Streptococcus pyogenes, staphylococcus aureus, and albus are also found in the inner layer. They cause secondary infection in the lymphatic glands, lungs, spleen, and kidneys. Inasmuch as the fibrin is deeply attached to the mucosa, its removal is difficult and is attended by hemorrhage.

The pseudomembrane due to cocci is feebly attached and easily removed, while the mucous membrane is red and shiny and does not bleed.

Diphtheria—bacteriologic diagnosis: This may be made by smearing a portion of the false membrane, or some of the mucopurulent secretion, upon a cover-glass, staining it with methylene-blue and placing it upon a glass-slide and examining it with a microscope for the Klebs-Löffler bacillus. This method is rapid, but not certain to reveal the bacilli even when present in large numbers. A slower but much surer way is to make a culture of the mucus obtained from the diseased membrane on dried blood-serum, such as is used in the health departments of our large cities. The bacilli develop more rapidly and more characteristically on blood-serum than on other mediums.

The *gross appearance* of the Klebs-Löffler bacillus culture is that of pin-head whitish-gray specks, with regular outline and dry surface. The centre of the colony is darker and more opaque than the edges. The colony develops in twelve to eighteen hours, which no other membrane-producing organism will do.

The streptococcus develops white pin-point specks after twenty-four hours.

The staphylococcus growth is larger, flocculent or snow-white, thin, and iridescent at the edges.

Diphtheria—clinical diagnosis: It is rare under one year of age. It is most often located on the faucial tonsils; and in the nostrils next most frequently. It may be in the nostrils and on the fauces at the same time, in which event the mortality is high. This is accounted for by the fact that the nose affords a large and rapid absorbing surface whereby the toxin enters the blood in large quantities. In nasal diphtheria the discharge is fetid, sanious, and irritating to the upper lip, which becomes excoriated and encrusted. Epistaxis is a common occurrence in this type. The sloughing which causes the hemorrhage exposes a surface through which rapid infection may occur and cause great prostration, pulmonary œdema, albuminuria, etc. The infection may gain entrance to the cranial cavity and cause meningitis. Nasal diphtheria is usually a mixed infection. The Eustachian tubes, middle ear, internal ear, and eye are sometimes the seat of diphtheritic pseudomembrane. Periostitis and œdema over the mastoid are complications occasionally met with. Simple and granular conjunctivitis predispose to eye-diphtheria. The resultant opacity of the cornea may cause partial or complete blindness.

Inflammation of the cervical and submaxillary glands is an early and significant sign in diphtheria. They may be swollen before the appearance of the membrane. If the case is one of pure diphtheria the cervical glands alone are swollen; if the infection is a mixed one both the cervical and submaxillary glands are enlarged and tender; and if the infection is from cocci, as in scarlatina, the submaxillary glands only are affected.

The diphtheritic membrane appears in varying colors according to the stage in which it is seen and the type of infection. It ranges from a white to a gray, yellowish-green, brown, and even to black, according to the quantity of pus and blood mixed with it.

Diphtheria—visceral complications: The bronchi are sometimes hyperæmic and œdematous. Broncho-pneumonia may develop as a result of coccal infection in the mixed type. Heart-clots occasionally develop and muscular degeneration of the cardiac muscles, rendering the action of the heart weak and irregular. The liver, spleen, and kidneys are enlarged.

The tubules of the kidneys become inflamed and casts are thrown off with the urine.

Diphtheria—Symptoms.

They vary with the severity of the disease, type, and location of the area of infection, age of patient, and the stage of the disease.

The **period of incubation** ranges from two to five days. Symptoms are not always present during this period; but when present they consist of general malaise and slight chilliness.

At the end of the incubation period the temperature ranges from 99° to 103° F. It usually remains about the neighborhood of 100° F. Great pyrexia is not a characteristic feature of diphtheria. Low temperature with profound depression is a sign of great toxæmia and is of grave import.

The **onset of the disease** is attended by slight elevation of temperature, quickened pulse, slight chills or chilly sensations, dry throat, discomfort or pain upon deglutition, malaise, stiff neck, aching in bones, headache, and other febrile phenomena. Any or all these symptoms may be very slight or wanting. By the end of the first day or beginning of the second there are lumbar and dorsal pains, the skin is flushed, the pulse quick and perhaps feeble, the cervical glands enlarged and tender, the urine scanty and high colored, the patient dull and unobservant.

The **malignant type** is usually unattended by prodromal symptoms. It begins with a heavy chill, vomiting, and sometimes convulsions. The temperature is slightly elevated and disappears on the second day. The heart is feeble, irregular, and very rapid, the skin a dusky gray, the urine scanty or entirely suppressed, and there is delirium of the low muttering type. Food is refused more on account of indifference than pain or difficulty attending the act of deglutition. Another symptom of importance is the absence of the tendon-reflex.

The *tendency to laryngeal diphtheria* is not so pronounced as in the milder types. Death is apt to occur by the second or fourth day. If death does not occur by the second to the fifth day, laryngeal stenosis is liable to take place.

The **pseudomembrane** at the beginning of the *first day* appears as a delicate pearly gray film, slightly elevated above the surface of the mucosa, which is swollen and dark red in color. If the diphtheritic membrane is limited to one side, the catarrhal turgescence of the mucosa will also be unilateral.

At the beginning of the *second day* the membrane, which on the first was in patches, is now extended until it covers all or nearly all of the tonsil. Its color is yellowish and opaque, and the surface presents a soft, velvety appearance. It stands out more prominently from the surface of the mucosa, giving the impression of considerable thickness. Mucopurulent secretion is noted about the edges, separation from the underlying mucosa having begun. It may come away in fragments or *en masse.*

By the *third day* the membrane is brownish or blue in color owing to the admixture of blood. It is more elevated from the surface and separation is complete. The temperature subsides, and if it should become elevated again reinfection has occurred or a new area has become involved.

If reinfection does not occur, the febrile movement will have entirely subsided by the end of the fourth day. Careful attention should be given the case for a week longer, as a recurrence may take place or some serious complication arise.

The **tongue** on the second or third day of diphtheria becomes dry, furred, and brown, while the breath is fetid.

Up to the second and third day the **chief danger** is from *septic poisoning.* After the second day it is from *laryngeal stenosis,* although septic poisoning and other conditions may also lead to a fatal termination.

Invasion of the larynx is marked by hoarseness or aphonia, which is followed some hours later by dyspnœa, both inspiratory and expiratory, although inspiration is more difficult. Subclavicular and abdominal depression are marked. The laryngeal invasion is attended by a sudden increase in the temperature. The membrane may be expelled in part or as a cast on the third or fourth day, and the process of resolution or that of the reformation of the pseudomembrane may now take place.

Diphtheria—prognosis: This varies with the epidemic and the stages of the same epidemic. The beginning of an epidemic is often mild, rapidly becoming more virulent, and finally subsiding until all virulence is lost.

Climate and *season* influence the prognosis, warm weather rendering it more favorable. *Early depression* indicates a profound toxæmia and renders the prognosis very grave. *Nasal diphtheria* is attended by rapid absorption of toxin. *Laryngeal stenosis* is always a grave symptom, but treatment often succeeds in averting a fatal issue. *Paralysis of the cardiac* and *respiratory muscles* may increase the gravity of what is otherwise a mild case. *Nephritis* adds about 2.5 per cent. to the mortality-rate. *A high temperature* should be regarded as an evidence of complications of a serious nature. *Meningitis* and *visceral complications* add to the gravity of the disease, and should render the prognosis guarded, even in mild forms of diphtheria.

Diphtheria—Treatment.

Although the introduction of *antitoxin* as a remedy has effected a revolution in the treatment of diphtheria, other remedies of recognized value should be given with as much care and discretion as though antitoxin had never been heard of.

The treatment may be studied under the following divisions: (1) general or systemic, (2) local, (3) antitoxin, (4) surgical, and (5) sanitary.

Diphtheria—general or systemic treatment: The *purpose* of general treatment is to promote the elimination of the toxin and to increase the vital power of the cells of the body so that they will resist the toxin until the course of the disease has expended itself.

Among the *remedies* which promote the elimination of the toxin calomel stands at the head. It should be administered in full doses at frequent intervals so as rapidly to obtain its effect. A preëxisting diarrhœa should not prevent its use. Turpentine in milk has advocates, and it will at least stimulate the excretions through the kidneys. Alcohol in liberal quantities is of undoubted value in cases in which there is great depression due to toxæmia. Children of a few years of age may be given 8 to 10 ounces daily without danger of intoxication, and with the most happy results. The carbonate of ammonium adds to its effect. The tincture of iron in glycerin (1 drachm to the ounce), given in teaspoonful doses every two hours, has a decided influence upon the local as well as upon the general condition of the patient. It adds to the cardiac-, respiratory-, and blood-power, enabling the system more effectually to resist the toxins already in the circulation, and perhaps to check their production.

General tonics should be given at the commencement of convalescence in order to promote assimilation and digestion. Water should be given freely, as there is need of the fluid to dilute and carry off the toxins through the excretory organs.

Diphtheria—Local Treatment.

Local applications of *lactic acid* in one-fourth to one-half strength to the diphtheritic membrane by means of a cotton-wound applicator facilitate the exfoliation of the pseudo-membrane from the mucosa.

Hydrozone has the double effect of promoting the separation of this false membrane and diminishing the virulency of the microbic action. It should be applied with a swab or spray (Fig. 116) every one or two hours.

Insufflation of sulphur is of especial value in those cases in which coccal infection is predominant.

The *normal salt solution* (0.3 per cent.) or *boric acid* in solution should be used every hour in the mouth, nose, and fauces by means of a syringe. This is preferable to the use of gargles, as they are usually inefficient in children. This is especially true in diphtheria attended by marked depression.

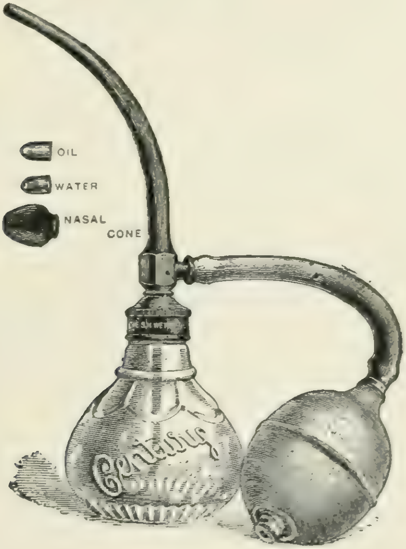
The following formula prescribed by Löffler is a *local remedy* of considerable value :

℞. Menthol,	10 parts.
Toluol,	26 "
Sol. perchloride of iron, fort.,	4 "
Absolute alcohol,	q. s. ad 100 " —M.

Sig. To be applied locally once or twice daily.

When diphtheritic membrane has extended from the fauces into the nose an effort should be made to remove it, as absorption takes place very rapidly, quickly producing profound toxæmia. The mucosa of the nose should first be cocainized to reduce the turgescence. It should next be irrigated with a syringe applied to the anterior nares. The head of the patient should be thrown forward over a basin so that the solution (normal salt or boric acid) will make its exit through the opposite nostril. The diphtheritic membrane should be carefully removed under reflected light and the persulphate of iron applied to the denuded surface.

FIG. 116.



Century atomizer.

Inhalations of the vapor of slaked lime is an old and valuable remedy in laryngeal diphtheria. It should be administered as follows: A temporary wigwam or cone-shaped tent should be constructed by the use of sheets. A chair, and a wooden vessel of two to three gallons capacity half filled with water, should be placed within the tent. Two or three pounds of fresh lime should then be thrown into the vessel, where it soon throws off steam charged with small particles of the lime. The patient should be placed upon the chair or held upon the lap of the nurse within the wigwam for five or ten minutes. This procedure should be repeated every two or three hours. In this way the diphtheritic membrane may be caused to separate from the trachea and bronchial tubes and be expelled in fragments or *en masse.* Turpentine may be added to the lime vapors with advantage.

Antiseptic alkaline sprays are of value in mild cases. They should be used in the fauces every hour.

Diphtheria—Antitoxin Treatment.

The theory: The blood-serum of animals rendered immune by the hypodermic injection of pure cultures of the Klebs-Löffler bacillus has the property of preventing and sometimes curing an animal subjected to the diphtheritic poison. It has the same power, in a more limited degree, in the human organism. It controls the pathogenic action of the micro-organism, or it neutralizes the toxin resulting from its presence. Its action is also explained as being due to rendering the cells of the body tolerant of the toxin. When the disease has gone on to the stage of profound depression there is no response to the administration of the antitoxin.

The **quantity necessary** varies with the individual, the epidemic, the part infected, and the age of the patient. In a general way it may be stated that the dose for a child under two years of age should be from 600 to 1000 units; while 1500 to 2000 units should be given to patients more than two years old. A prophylactic dose is from 600 to 800 units. The dose may be repeated in twelve to twenty-four hours if improvement has not set in. A third dose may also be given without danger in unyielding cases. The effect of the antitoxin is manifested by the subsidence of the toxic symptoms and the disappearance of the pseudomembrane.

Effect on rate of mortality: If it is administered during the first twenty-four hours, the death-rate is about 12 per cent.; if given during the second twenty-four hours, it is about 25 per cent.; and if given during the third twenty-four hours, it is about 35 per cent. Hence the importance of early administration is quite evident.

The **sequelæ** usually following diphtheria are in no way influenced by antitoxin.

The **mode of administering** antitoxin is by hypodermic injection, the subscapular region being most often selected for the purpose.

Diphtheria—Surgical Treatment.

Indications: When there is progressive asphyxia, signalized by aphonia, dyspnœa, stridor, and recession of the chest and abdominal walls either *intubation* or *tracheotomy* should be done without delay.

The **dangers** attending either of the procedures is so small as compared with those of non-interference that the latter should not be allowed to weigh against the former.

Diphtheria—Intubation.

The **choice** between intubation and tracheotomy should be determined by the age of the patient and the wishes of the parents. Until the fourth year intubation gives a mortality-rate 10 per cent. lower than tracheotomy. From the fourth to the fifth year the results of the two operations are about equal. After the fifth year tracheotomy seems to give the most favorable result.

Method of introducing the tube: The patient should be taken on the lap of the nurse, his head held firmly against her left shoulder with the left hand while his hands and arms, folded in front, are held with her right hand and arm. If need be, a binder or sheet may be wound about the child, thereby enabling the nurse to hold him with greater ease and security. His legs should be fixed firmly between the knees of the nurse. An assistant stands behind, introduces a mouth gag upon the patient's left side, steadying the head of the child between his hands.

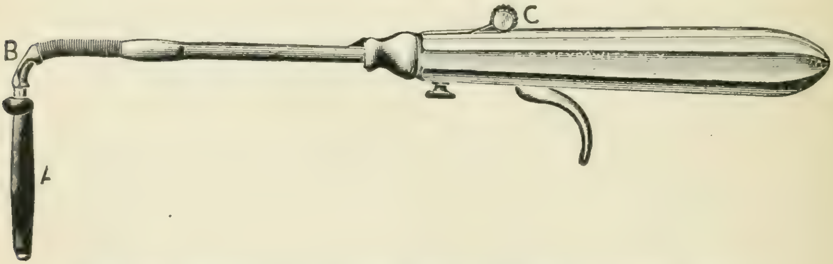
The surgeon should stand or sit in front of the child and make a hasty laryngoscopic examination to determine if there is a fragment of membrane lying over the glottic fissure.

This sometimes happens and its removal with a swab or forceps obviates the necessity of intubation or tracheotomy.

If no such obstruction exists the index-finger of the left hand is introduced into the mouth and pharynx, catching the right side of the epiglottis and holding it against the root of the tongue. If the epiglottis cannot be felt (it is quite small and soft in young children) the tip of the finger should find the arytenoid cartilages and use them as landmarks. If the finger is over the centre of the epiglottis it interferes with the introduction of the tube.

The tube is fixed upon the introducer (Fig. 117) and passed into the mouth with the right hand, the handle of the introducer being placed against the chest of the child and gradually raised as the tip of the tube passes back of the dorsum of the tongue.

Fig. 117.



Intubator with a tube in the proper position for insertion into the larynx: A, intubation tube; B, fork pushing tube away from obturator; C, knob which, when pushed forward, causes the fork to strike the head of the intubation tube.

When the tip of the tube has reached the tip of the finger holding the epiglottis in position, the handle of the introducer should be suddenly elevated and slightly drawn forward, the tube being made thereby to pass *downward and forward* along the palmar and thumb surface of the finger into the larynx. If the handle is not thus suddenly elevated and drawn forward the tube will enter the œsophagus.

As the patient is in great distress the whole procedure should be done with precision and speed.

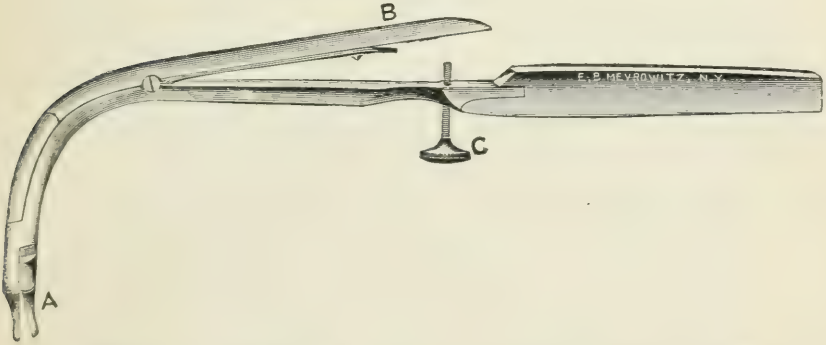
Before introducing the tube it should be threaded through its shoulder by a strong silk or linen thread about sixteen inches long, the ends of which are tied, forming a loop. This should be hooked over the little finger of the left hand ready for use should the tube enter the œsophagus, or need to be removed for any other reason.

When the tube is properly adjusted the thread should be cut and removed, the index-finger being introduced and held against the shoulder of the tube to prevent its withdrawal.

If the tube is too small it will be coughed up.

A scale is provided with each set of instruments, so that even the inexperienced can select a tube of the proper size. Five tubes of various sizes are usually furnished with each set.

FIG. 118.



Extubator: A, the jaws partly open; B, lever, pressure on which in a downward direction opens the jaws; C, screw which regulates the extent to which the lever may be depressed and the jaws opened.

The **objections to intubation** are: (*a*) the liability to cough up the tube and the necessity of calling the physician to reintroduce it; (*b*) it sometimes interferes with deglutition: liquid food can usually be swallowed. If food enters the trachea the patient should be made to lie upon the stomach and take the food through a tube; (*c*) The opening of the tube being small it sometimes becomes obstructed by false membrane, thereby necessitating its immediate removal. This is sometimes accomplished by the violent efforts of the child.

The **tube should be left in place** until convalescence is well established, as evidenced by the disappearance of: the faucial membrane, the great depression, and the feeble rapid pulse.

Its **removal** is accomplished with the extubator shown in Fig. 118. The tip should be introduced into the mouth of the tube, guided by the index-finger of the left hand. It is then widened by pressure upon the lever B, and the tube removed by reversing the movements used in its introduction.

Diphtheria—Tracheotomy.

The **indications for tracheotomy** are much the same as those for intubation, the chief difference being the age of the patient. After the fifth year tracheotomy affords a somewhat better mortality-rate than intubation. Other points in its favor are (*a*) the ability to get lower into the trachea than can be done by intubation; (*b*) the tracheal diphtheria can be treated locally through the wound; (*c*) deglutition is not disturbed; (*d*) respiration is freer; (*e*) the inner tube can be removed and cleansed frequently by the nurse without calling for the surgeon; and (*f*) should it become obstructed by false membrane it can be removed and freed from it without delay or prolonged danger.

The **objections to tracheotomy** are that it is a surgical operation, and excites the prejudice of the parents; it is more apt to be followed by bronchopneumonia than is intubation, on account of the loss of nasal respiration; intubation is much more easily performed, and is therefore of more universal application.

Intubation is much more commonly practised on account of its apparent simplicity. All physicians are not prepared or willing to perform tracheotomy. The probability of death is so great under any mode of treatment that physicians hesitate to perform what, to the minds of the parents, might be considered the cause of death.

The objections to tracheotomy are largely sentimental, but they are none the less potent on that account.

The **death-rate** in those cases *actually requiring* and receiving intubation or tracheotomy is about 80 per cent. In other words the percentage of recoveries is about 20 per cent.

Statistics can be made to show a much greater percentage of recoveries by taking into account all the cases intubated by enthusiastic supporters of this mode of treatment when only slight laryngeal stenosis is considered a sufficient cause for intubation.

While it may be a wise procedure to intubate in slight laryngeal stenosis, it is not just to compare the results with those of tracheotomy, an operation only resorted to as a last resort.

As all works on general surgery describe the various **methods of performing tracheotomy** they will not be given in this brief work.

A few suggestions as to the **after-treatment** only will be given. The wound about the tracheotomy tube should be covered with antiseptic gauze. The opening in the tube should be lightly covered with one or two thicknesses of gauze to filter the dust from the inspired air. The atmosphere of the room should be kept moist by steam vapors to provide moisture for the inspired air. The temperature of the room should be maintained *constantly* at about 75° F. The inner tube should be removed and cleansed by the nurse every two hours. The entire tracheotomy tube should be removed every twenty-four to forty-eight hours and another of the same size introduced in its place. Warm antiseptic sprays may be used from time to time through the tubes to thin the mucous accumulations in the trachea.

Diphtheria—Sanitary Treatment.

The sanitary treatment of diphtheria in the home should consist of the following:

Isolation: The sick chamber should be one that can be isolated as much as possible from the other parts of the house.

Sunlight: It should be one exposed to the sunlight, preferably on the southeast or southwest corner of the house.

Ventilation: The room should be thoroughly ventilated all the time. It may be necessary to provide an extra amount of heat on account of the free ventilation.

All **rugs and hangings** should be removed from the room.

The **linen**, bedding, napkins, and handkerchiefs should be thrown immediately after use into a solution of mercuric bichloride and allowed to remain a few hours before washing.

The **expectoration** should be received into a vessel containing a solution of the bichloride of mercury.

Attendants: Only the nurse, or member of the family acting as nurse, should be allowed in the room. She should not mingle with the rest of the family, and should take long walks in the open air.

After convalescence has been established for seven to ten days the patient may be allowed to mingle with the family and resume his former place in child life.

The room, and perhaps the house, should be treated according to the method adopted by the **health board.**

NEOPLASMS OF THE PHARYNX.

Fibroma of the Nasopharynx.

Etiology: This is a firm fibrous growth arising from the base of the sphenoid bone and spreading to adjacent tissues and cavities, more especially the nose. It springs from apparently normal tissue; and occurs in persons who are otherwise healthy. Males seem to monopolize the disease, but few cases occurring in females. Age exerts a decided influence, it being rare under ten and over twenty-five years. Puberty marks the commencement of most cases. The growth is spontaneously arrested at about the age of twenty or twenty-five years.

Fibroma of the nasopharynx—symptoms: The early symptoms are those of slight irritation and nasal stenosis. As it progresses these become more pronounced. The extension and development of the tumor are usually rather slow, but may be quite rapid. Headache and aprosexia are usually present, as in all types of nasal stenosis. Frog-face or a wide nasal bridge becomes a prominent symptom. As the neighboring cavities are invaded pain becomes a symptom of importance. Hemorrhage may become a grave complication. The surface of the growth is interlaced by thin-walled vessels, which probably accounts for the hemorrhage. Upon examination with the rhinoscopic mirror a smooth, rounded tumor of a pale pinkish color is seen. Sometimes it is of a deep red or purplish color.

Diagnosis: Fibroma is firm, dense, and elastic to the touch, while sarcoma is soft and yielding. Both bleed upon slight mechanical irritation. The pain in sarcoma is apt to be referred to the ear. It may be differentiated from fibropolypus by its firm, broad attachment, while the fibropolypus is pendulous and easily moved. The fibropolypus is also much softer to the touch.

Prognosis: As there is no limit to the growth it may invade vital parts and become quite a grave disease. The tendency to severe hemorrhage also adds to the gravity of the condition by impairing the health or causing death. If it is early recognized and properly treated the prognosis is quite favorable.

Fibroma of the nasopharynx—treatment: *Injections* of the chloride of zinc in saturated aqueous solution, repeated at intervals of two or three days for a fortnight or more, has occasionally been successful. Acetic acid has been used in the same manner.

The *knife* is objectionable as its use is attended by serious hemorrhage.

Electrolysis has been used with about the same success that has attended its use in fibrous tumors elsewhere. It is not a certain remedy, inasmuch as the growth is dense and fibrous and does not contain the fluids the presence of which is necessary to the best action of electrolysis.

Method: An electrode with several pendant needles should be attached to the negative pole of a powerful galvanic battery. The positive electrode should be a large sponge (one foot square), and it should be placed upon the abdomen. The needles of the negative pole should be thrust deeply into the growth through the mouth. The electric current should now be gradually turned on by means of a finely graded rheostat until the milliampèremeter registers from 25 to 50 ma. Great caution should be exercised, as the growth is near the brain and syncope may result from a too sudden increase in the amount, as well as from an excessive amount, of current. If the growth were remote from the brain, 100 to 300 ma. might be advantageously used. The objections to this mode of operating are (*a*) the danger of electric shock to the brain; (*b*) its uncertain action; (*c*) it may need to be used more than once; (*d*) the unreliability of most medical electric instruments; and (*e*) the lack of scientific knowledge of electrotherapeutics among the majority of medical men.

The *cold-wire snare* and *écraseur* may be used, but the liability to hemorrhage is so great that their use should be undertaken with caution. A single case in my own experience emphasized the danger of this method. The hemorrhage was so sudden and profuse that death was imminent for more than two hours.

If the growth is large it may become necessary to make a temporary resection of one of the superior maxillæ to effect its removal.

Fibrous Polypus of the Nasopharynx.

Occurrence and structure: This is more rare than fibroma. It starts in the vault of the pharynx and, like fibroma, extends into the nose. The *pharyngeal* portion is more *fibrous*, while the *nasal* extension is more *myxomatous*, in structure. Unlike fibroma it does not distort the neighboring bones and cavities, nor is it attended by hemorrhage. About two-thirds of the cases occur in females, between the fifteenth and thirtieth years.

The **prognosis** is quite favorable under treatment. It is less apt to recur after removal than simple polypus.

Diagnosis: Articulation is impaired as the uvula and velum loose their mobility. The tumor hangs free in the nasopharynx and may sometimes be seen projecting below the soft palate.

Fibrous polypus of the nasopharynx—treatment: The growth may be seized through the mouth with a strong pair of forceps and twisted from its attachments. Damage, however, may be done to other structures by this procedure. If it is attached to the mucosa and periosteum of the body of the sphenoid, the bony walls of which are sometimes quite thin, evulsion may fracture the thin plate of bone and expose the sphenoidal sinus.

The *snare* is a safer instrument to use in these cases. Some ingenuity may be required to engage the growth in the loop.

Thornwaldt's Disease.

Definition: This is a condition in which there is a chronic inflammation of, and remnants of partially atrophied, *adenoids*. The thickening on the posterior wall of the nasopharynx is in the form of a ridge on either side of the median line. Pus accumulates in the groove thus created, becomes inspissated, and forms a central crust which is characteristic of this condition. It may be regarded as a postrhinitis sicca.

The **treatment** should be addressed to the attendant rhinitis; and the complete eradication of the fibrous adenoid ridges with the curette.

Chondroma of the Nasopharynx.

Symptoms: These are much the same as those of fibroma, except that the hemorrhage is absent. Nasal stenosis, aprosexia, headaches of long duration, and syncope may occur during the progress of the disease. Great distortion of neighboring bones and cavities occurs, as in fibromata. To the touch the tumor offers a firm, hard, resisting surface. It is not so hard but that a needle may be thrust into it. With the postrhinoscopic mirror it appears as a smooth, whitish-pink tumor.

Chondroma of the nasopharynx—treatment: Nothing short of a radical surgical operation will be effective. Langenbeck's operation for the temporary removal of the superior maxilla is the best method of exposing the growth.

Sarcoma of the Nasopharynx.

Occurrence: It is extremely rare. Unlike sarcoma in other parts of the body, it seems to occur with almost equal frequency at any time between the ages of one to fifty years. It occurs most frequently in males.

Sarcoma of the nasopharynx—symptoms: They are about the same as those of fibroma, except the hemorrhages are not so serious. There is an offensive discharge which is probably septic and accounts for the early disturbance of the general health. As the disease advances there are difficult deglutition, dyspnœa, pain in the ear, impaired hearing or complete loss of hearing. Objectively the tumor is soft and elastic, bleeds easily upon digital examination, unlike fibropolypus. The glands of the neck are not enlarged as they would be in carcinoma. Sight may be destroyed by pressure upon the optic nerve in its passage through the sphenoid, but both it and hearing may return after the removal of the tumor. There is a tendency to recurrence after removal.

The **treatment** should be about as for fibroma and chondroma.

Carcinoma of the Nasopharynx.

Symptoms: In its early stage it gives rise to the same clinical phenomena as sarcoma. There is early glandular enlargement, which persists throughout the progress of the disease.

As it is a fatal malady, the **operative treatment** will not be considered in this work.

THE UVULA.

Malformations of the Uvula.

Varieties: Bifid Uvula; Complete or Partial Absence of the Uvula.

Symptoms: Cough and irritation in the pharynx sometimes occur from the presence of a bifid uvula.

The **treatment** consists of removing the mucous membrane on the opposed surfaces of the divided uvula and bringing them together with catgut sutures.

Ulceration of the Uvula.

Occurrence: This is rare as a primary condition, and is characterized by pain upon deglutition. The ulcer is usually upon the posterior surface, and can only be seen with the aid of the laryngeal mirror. It may be the cause of a supposed postnasal catarrh.

The **treatment** should consist of the application of the solid stick of the nitrate of silver or the actual cautery.

Mycosis of the Uvula (Leptothrix).

Here, as in tonsillar mycosis, there are the ivory-colored, pointed, and elevated patches of leptothrix.

Acute Inflammation, or Œdema of the Uvula.

The **etiology** is very similar to that of acute tonsillitis, the rheumatic diathesis being a prominent factor.

The **symptoms** are those of slight obstruction, pain upon swallowing, and a tickling cough. A general pharyngitis is often associated with it.

Acute inflammation of the uvula—treatment: During the acute stage it should consist of scarifications and alum or zinc sulphate washes. After the subsidence of the acute stage it may be necessary to amputate the uvula on account of the elongation. When thus elongated it gives rise to a distressing dry cough.

Relaxation of the Uvula.

Occurrence: As this condition constitutes 90 per cent. of the affections of the uvula requiring treatment it will be given more space than its importance seems to warrant. It is a condition, however, of considerable interest to the laryngologist on account of its frequent occurrence in singers and public speakers. It is commonly associated with inflammation of the pharynx, and more especially in those who strain the voice in singing or speaking.

Etiology: In its enlargement the vascular, glandular, fibrous, or muscular tissue may predominate.

For reasons not now well understood there is sometimes an entire absence of muscular fibres in the elongated uvula. Senility cannot account for it, as the absence is noted in all ages. The absence of muscular fibres accounts for the laxity in some cases. As most persons afflicted with chronic relaxed throat have at some earlier period had diphtheria, scarlet fever, influenza, etc., the muscular atrophy may be due to paralysis arising during such attacks.

Relaxation of the uvula—symptoms: Coughing paroxysms occur when the general tone of the system is lowered—*i. e.*, of mornings and evenings. There is a tickling sensation in the throat with more or less discomfort and pain. Muco-gelatinous pellets are sometimes thrown out during the paroxysms. The expectoration is occasionally streaked with blood, which alarms the patient, as he suspects tuberculosis. This suspicion is still further confirmed by the prostration following the coughing.

A young man once came to consult the author concerning what he supposed was incipient phthisis. Examination of the chest was negative. Inspection of the throat revealed a relaxed uvula. This was amputated and all the alarming symptoms rapidly disappeared.

Singers are particularly annoyed by this condition as it incapacitates them from pursuing their chosen profession. The special symptoms in singers are pain and discomfort after using the voice, loss of brilliancy, uncertain register, impaired range, and unsteadiness of tone. In time hoarseness supervenes, thereby completely destroying the singing voice.

I must again quote Lenox Browne, *The Throat and Nose*, fifth edition. His direction for **objective examination** of the fauces are of exceptional value, and are as follows:

"1. Direct the patient to open the mouth without taking a breath, and the relaxed uvula, which, if in a normal condition, should on inspiration be retracted, will be seen to be lying on the tongue.

2. Should the palate not drop by the patient thus holding the breath, direct him to breathe out through the nostrils, which will have the result of relaxing the palate, and the length of the uvula can be estimated.

3. Let the patient then breathe in deeply through the mouth or strike a high note, and it will be seen that the uvula is not entirely drawn up, owing to paresis of tensor palati ; or that the uvula goes up in wrinkles, partly from the same cause and partly from the excess of relaxed tissue.

4. Remember that the amount of relaxation depends on the relation which the length of the uvula bears to the arch of the palate.

5. In those cases in which, observing all these precautions, the uvula does not appear to be relaxed, and yet there is no

other reasonable cause for the symptoms, observe carefully the edges of the curtain of the soft palate, and they will be seen to be thin, white, and quite translucent, and almost to flap about with the respiration."

Relaxation of the uvula—treatment: The classical treatment has long been astringent gargles of alum-water and solutions of tannin. Astringent lozenges with eucalyptus, cubebs, chlorate of potash, guaiacum, and menthol are now more commonly employed. After using one or more of these remedies for a few days the amount of permanent elongation can be accurately estimated. If the elongation is still excessive a portion of the uvula should be amputated. The incision should be made in such a manner as to leave the wounded surface toward the posterior wall of the pharynx. The act of swallowing food will thus be free from discomfort and irritation. The operation may be performed by grasping the tip of the uvula with a pair of toothed forceps, drawing it forward and downward and amputating above the forceps with curved uvula-scissors; or it may be done with a uvulatome, an instrument resembling Mathieu's tonsillotome; it may also be removed with an electrocautery snare. A 5 per cent. solution of cocaine should be applied to the surface at intervals for a few minutes before resorting to either of the foregoing procedures.

THE TONSILS.

THE FAUCIAL TONSILS.

Foreign Bodies in the Tonsils.

The tonsils when enlarged present a surface with numerous pits or lacunæ. They lie between the anterior and posterior pillars of the fauces, hence they present favorable conditions for the lodgment of foreign bodies, such as fish bones, sharp splinters of bone or wood, pins, needles, and other sharp substances.

The patient should be directed to relax his throat as much as possible during the examination, as otherwise the tense pillars of the fauces may conceal the foreign body from view.

Faucial Tonsils—Acute Inflammations.

General description: Under this caption will be considered what is sometimes described as several distinct diseases, *i. e.* (*a*) *acute superficial tonsillitis;* (*b*) *follicular tonsillitis;* and (*c*) *parenchymatous tonsillitis.*

These conditions are much the same except in degree, the nature of the exudate, and the point of greatest inflammation. Clinically the various conditions are often associated at some stage of the process and the symptom-complex can be best studied as an entity.

More than 50 per cent. of all faucial and pharyngeal diseases are of the inflammatory type. When the crypts, of which there are ten to fifteen in each tonsil, are chiefly involved they become distended with epithelial cells, leucocytes and microörganisms. Streptococci and staphylococci are the ones chiefly associated with this type of tonsillar inflammation.

Etiology: The parenchymatous inflammation may be attended by hypertrophy of the tonsillar tissue, especially after repeated attacks. In children it seems to be a histopathologic law that slight irritations may cause hypertrophy of the lymphoid tissues. Such irritations also may cause it as arise during a cold, scarlet fever, measles, influenza, diphtheria, and other exanthematous fevers. It should be remembered, however, that all children who are thus exposed do not have enlargement of the tonsils. There must be some other condition which determines the hypertrophic process. The strumous and rheumatic diatheses undoubtedly play an important rôle in this connection. In other words a chronic disturbance of the digestive and metabolic processes predisposes to tonsillar inflammation. Just what the primary cause may be is, perhaps, not now known. Heredity should, however, be considered in studying the etiology of tonsillar diseases, as it undoubtedly has an influence.

Tonsillitis is sometimes epidemic over considerable areas. Exposure to wet and cold, the sudden changes of temperature, insufficient clothing and protection to the feet, are sufficient to cause an attack of acute tonsillitis in many individuals.

The disease is most common at the period of adolescence and young adulthood, although it may occur at any age.

Faucial tonsils—acute inflammations—symptoms: These vary with the degree, type, and location of the focal points of inflammation. In the *simple diffused form* there is slight swelling and redness accompanied by a feeling of discomfort upon deglutition. The pain is often described as a prickling one, as from the presence of a fish-bone or other small sliver in the fauces. When the tonsillar crypts are filled with epithelial cells, leucocytes, and microörganisms there is not so much pain, but there is considerable enlargement.

If the crypts are lined with a fibrinous exudate, the symptoms are apt to be much more severe. The rheumatic diathesis is particularly manifest in this form of tonsillar inflammation. The parenchyma of the tonsil, together with the contiguous structures—*i. e.*, the pillars and uvula—are often inflamed and œdematous. This *fibrinous type* was formerly often mistaken for true diphtheria. The pseudomembrane is not, however, adherent to the surface of the tonsil as it is in diphtheria. A bacteriologic examination of the tonsillar secretion will aid in making a correct diagnosis.

Faucial tonsils—treatment of acute inflammations: All forms of acute tonsillitis, including inflammations of the areolar tissue surrounding the tonsils, may in many instances be aborted by the timely administration of the *tincture of aconite* in 1-minim doses every hour until there is tingling in the fingers or a dry prickling sensation in the fauces. To be effective it should be given during the first twenty-four hours of the disease.

As tonsillitis is often associated with the rheumatic diathesis, the *salicylate of soda* should be given in 3-grain doses every hour until there is marked improvement in the symptoms. The internal administration of the *carbonate of guaiacol* in 5-grain doses every three hours is a remedy of considerable value in the exudative type.

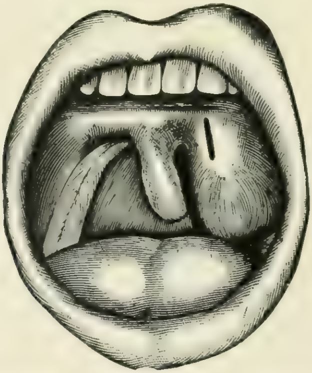
Guaiacol, with an equal amount of olive oil; or a 50 per cent. aqueous solution of the nitrate of silver, is a most excellent *local application* in the acute stage of the superficial inflammation. The application of the nitrate of silver in this strength is attended by some danger, as a single drop of it flowing down upon the larynx would excite most violent laryngeal spasms. Extreme care should be taken to remove the excess of fluid from the cotton-mop before applying it to the surface of the tonsil. Solutions of this strength are of no value in the later stages. In the acute stage, however, they act as specifics. In the author's experience only one or two applications have been required to abort the inflammation. The effect is to produce rapidly local anæsthesia and blanching of the mucous membrane.

Other local remedies of value are solutions of the salicylate, bicarbonate and chloride of sodium, and menthol. The menthol should be applied in 10 per cent. oily solution with an atomizer.

Peritonsillar Abscess; Quinsy.

Symptoms: If the inflammation is chiefly centred in the loose connective tissue surrounding the tonsil (Fig. 119) the movements of the lower jaw are very much interfered with.

Fig. 119.



Peritonsillar abscess on the left side, including line of incision. (Coakley.)

The soft palate and uvula are œdematous and nasal respiration impaired. The secretions are profuse and stringy and may be composed of mucous or mucopurulent matter. The patient has a stupid, anxious cast of countenance, the mouth being open and the saliva driveling therefrom.

The pus may point above and anterior to the tonsil, through the anterior pillar; or it may point low through the posterior pillar into the pharynx. Upon *inspection*, one or both sides of the fauces will be found to be very much swollen and œdematous, and covered with heavy mucus which is mixed with epithelial cells, cocci, and leucocytes.

Quinsy—treatment: The tincture of aconite if administered in the early stage, as given under acute inflammation of the tonsils, may abort the attack before the formation of pus occurs. Bosworth recommends that the patient be provided with a saucer of the bicarbonate of soda into which he should dip his moistened finger at frequent intervals and plaster it on the inflamed tonsil. He claims that it reduces the amount of pain and exerts a favorable action on the course of the disease. If marked depression is present the tincture of iron and alcoholic stimulants should be given.

When the disease has progressed to abscess-formation the point of greatest fluctuation should be located and freely incised with a bistoury. It usually points above and anterior to the tonsil, as shown by the straight line in Fig. 119. A small, pointed bistoury should be used, making a free incision parallel with the free border of the anterior pillar, so as to avoid cutting the fibres of the palatine muscles. The edges of the wound should be separated with a pair of forceps to facilitate the escape of pus. It is not best to wait for spontaneous rupture of the abscess, as it may burrow beneath the deep fasciæ of the neck and lead to serious complications.

Hypertrophy of the Faucial Tonsils (Chronic Enlargement).

Etiology: The chief *predisposing causes* of enlargement of the tonsils are the lymphatic habit and the strumous diathesis. Heredity undoubtedly exerts an influence. It is a disease peculiar to childhood, rarely having its beginning in adult life. With the passing of the period of adolescence the tendency to hypertrophy disappears. Sclerosis and shrinkage of the tonsillar substance begins and may continue until it is quite firm and gristly. Hypertrophy rarely begins after puberty.

The *exciting causes* are diphtheria, scarlet fever, measles, small-pox, and acute inflammations of the fauces.

In some cases no cause can be determined, hence it is said to be "idiopathic."

Morbid anatomy: Three general classifications may be made, viz.: (1) *True hypertrophy*, in which all the elements composing the tonsil are increased in size and number. The lymphoid nodules are particularly thus affected. (2) *Hyperplasia*, or sclerosis, in which the stroma or connective-tissue framework of the tonsil is increased. As the process advances the bloodvessels and lymphoid nodules are diminished in size. The tonsil, instead of being soft, as in the hypertrophic variety, is firm and unyielding to the touch. (3) The *lacunar type* is characterized by a ragged appearance, and has been called the "honey-combed" tonsil. There are normally from ten to fifteen crypts in each tonsil. They become enlarged from being distended by accumulations of epithelial cells, leucocytes, and débris. A probe can be passed into them for a half inch or more. In the lacunar or "ragged" tonsil they are sometimes found to communicate with each other. This may be due to the absorption of the intervening wall by pressure necrosis; or to a caseous degeneration of the lymphoid nodules, leaving cystic cavities where true parenchyma once existed.

Sometimes the pillars of the fauces are adherent to the tonsils and should be dissected loose before attempting the removal of the latter.

Chronic enlargement of the tonsils—symptoms: These are mainly those due to mechanical interference with the normal functions of the fauces, larynx, nasopharynx, and ear.

The muscles of deglutition are hampered in their action, and the child in consequence drinks considerable quantities of water with his food. Hyperaemia of the larynx is induced, and the voice may be husky, toneless, and easily fatigued. The tensor palati and levator palati muscles are obstructed in their action, and as they largely control the patency of the Eustachian tubes, the proper aeration of the tympanum is not accomplished. Catarrh of the tubes and middle ears is thereby developed. This may assume the suppurative type if virulent pus-cocci gain entrance into the tympanum.

Hypertrophy of the pharyngeal, lingual, and nasal lymphoid masses or tonsils is commonly associated with enlargement of the faucial tonsils. The nose is therefore obstructed by the postnasal adenoids and the intranasal tonsils (lymphoid nodules in the nasal mucosa), as well as by the catarrhal thickening of its mucous membrane.

As a *result* of the obstruction to nasal respiration the patient snores during sleep. The facial expression is somewhat dull, and the mind is often as dull as the face betrays. Aprosexia, or difficult attention, is a common symptom. The chest is often malformed, pigeon-breast being the usual type. This has been explained upon various hypotheses, most of which are entirely unsatisfactory. The mechanical hypothesis is inadequate and illogical. The true explanation is probably to be found in the struma and malnutrition that predispose to the lymphoid hypertrophy. In other words there is a common cause for both conditions.

The sense of smell and taste are sometimes impaired.

Objectively the tonsils appear as roundish masses projecting from between the anterior and posterior pillars of the fauces. In the hypertrophic type they are of a pinkish-red color, while in the hyperplastic type they are much paler. In the lacunar variety they appear as ragged masses or, if seen in the earlier stage, as swollen tonsils, with the crypts filled with an ivory-colored concretion.

Hypertrophy of the faucial tonsils—treatment: As the predisposing causes of hypertrophy of lymphoid tissue are the strumous and the lymphatic habits, *constitutional remedies*, as cod-liver oil and iron, are indicated. The food should be nutritious and easily digested. The clothing should be so regulated as to protect the child from the inclemency of the weather, as it is quite important that he should have an abundance of exercise in the open air.

Numerous *local remedies* applied to the tonsils for the purpose of causing them to shrink have been tried from time to time.

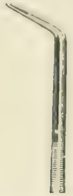
They, however, do no more than reduce the hyperæmia of the mucosa, the hypertrophic process being in no wise arrested.

The tincture of iodine has been applied to the tonsils and to the skin over the angle of the inferior maxilla with the vain hope of arresting the tonsillar growth.

Chemical caustics were formerly in vogue, but the electrocautery has superseded them.

Ignipuncture is performed as follows: 1. The tonsil is painted at intervals of one minute with a 10 per cent. solution of cocaine until local anæsthesia is produced. 2. The electrode (Fig. 120) should be a simple pointed one. 3. It should be placed against the tonsil while cold, and the current connected, so regulated previously as to bring the platinum point to a red heat. The electrode while thus heated should be plunged to the depth of half an inch into the tonsil. This procedure should be repeated three or four times at the same sitting.

FIG. 120.



Galvanocautery electrode.

The scar-tissue resulting from repeated ignipunctures causes the tonsil to diminish in size. This mode of treatment is to be condemned, as it often leaves the tonsils in such a ragged condition as to afford a lodging place for detritus and microorganisms.

The *lacunar variety* of enlarged tonsils may be treated as follows:

1. Remove the accumulations of epithelial cells and leucocytes from the crypts by squeezing the tonsils; or by means of a tonsil hook.

2. Cocainize the crypts of the tonsil with a cotton-wound applicator saturated with a 20 per cent. solution of cocaine.

3. Introduce the cautery tip heated to a cherry-red color, into four or five of the crypts at each sitting.

4. This procedure may be repeated at intervals of from four to seven days until all the crypts have been thus cauterized.

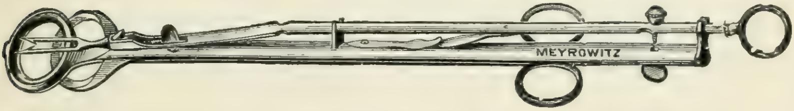
All the foregoing *local* methods of treatment are but makeshifts, to be used only when the following radical and more reliable methods are refused:

Tonsillotomy: It is commonly and very satisfactorily done with Mathieu's (Fig. 121) or Mackenzie's tonsillotome. Mathieu's instrument is a ring-knife cutting from behind forward, while the Mackenzie or spade tonsillotome cuts from before backward. The operation may be done as follows:

The *anæsthesia* may be local or general. Cocaine (10 to 20 per cent. solution) applied with a cotton-wound applicator at intervals of one minute will produce local anæsthesia in from five to ten minutes. The general anæsthesia may be produced by the administration of chloroform ; ether ; A. C. E. mixture ; Schleich's mixture ; bromide of ethyl ; or laughing-gas.

Of these ether is perhaps the least desirable, as it is irritating to the air-tract. It has been my own custom to administer the bromide of ethyl where general anæsthesia was desired. The child, if under seven years of age; should be held in the lap of an assistant whose legs should be crossed over those of the patient. The hands and arms should be held with the left hand and arm of the assistant while the right hand holds the head firmly against the right shoulder. If the patient is particularly unmanageable a sheet should be wound about him, thereby enabling the assistant to hold him with greater ease. The bromide of ethyl, which is put up in hermetically sealed tubes, should now be administered much after the same method followed in chloroform anæsthesia. As the anæsthesia lasts from two to three minutes ample time is afforded for the thorough removal of the faucial tonsils and postnasal adenoids which often coexist.

Fig. 121.



Mathieu's tonsillotome.

Introduce a mouth-gag.

The *tongue* should be held down with a tongue-depressor while the tonsillotome is placed over the tonsil and insinuated upon the same.

The *tonsils may be made to protrude* into and engage in the tonsillotome more readily if an assistant exerts pressure under the angle of the jaw.

The moment the tonsil *projects through the ring* of the knife, the blade should be forced through its substance and the operation thus completed.

The instrument should be reversed and the tonsil upon the opposite side removed in the same manner.

The patient should then be quickly thrown forward over a basin to prevent the blood from entering the œsophagus and trachea.

The *after-treatment* of the throat should be as for simple acute tonsillitis.

The tonsils may also be removed with **a cold snare.** This method is more practicable in adults than children. Enlarged tonsils in adults are almost always of the hyperplastic variety, in which the walls of the tonsillar arteries are held open after incision, by the firm, fibrous, noncollapsible tissue of the tonsil. In this class of cases either a dull tonsillotome or the snare should be used.

The **hot wire** is also used by some operators to diminish the liability to primary hemorrhage. Secondary hemorrhage is more apt to occur after removal by this method.

Another method of removing the tonsils has been devised by Dr. Pynchon, which he calls the **cautery dissection** of the tonsils. He claims that the results are so much superior to those of *tonsillotomy* that the difficulties of the method are more than compensated for. The steps of the operation are as follows:

Cocainize as for tonsillotomy, with the exception that the pillars of the fauces are also anæsthetized.

Seize the upper portion of the tonsil with a pair of long-toothed forceps, and begin dissecting with a suitably bent or straight cautery.

One half of the tonsil should be removed at the first sitting, the remainder in the course of ten to fourteen days.

Severe secondary hemorrhage has occasionally followed this procedure.

Hemorrhage is prevented or the probability thereof reduced to the minimum by the application of a 50 per cent. solution of the nitrate of silver to the wound immediately after the operation. The wound should be gently massaged with a cotton-wound applicator dipped in equal parts of the tincture of iron and glycerin after the second or third day.

The whole tonsil should rarely be removed at one sitting, as the danger of secondary hemorrhage is very much increased thereby.

The author's experience with this method of operating has been limited to a few cases, all of whom have been adults. This method of tonsillotomy is of special value in the sclerotic tonsil. By its careful use the danger of hemorrhage is minimized. It is of special value in the removal of enlarged tonsils of singers and public speakers, as it gives a cleaner result than any other method except enucleation.

This subject should not be dismissed without a final word of caution, namely, that cautery dissection is a dangerous procedure unless the operator is thoroughly familiar with the technique of the operation.

The **enucleation of the tonsils** with the finger is destined to receive more attention in the future than it has in the recent past. Adults with fibrous tonsils should be anæsthetized, the head hanging over the end of the operating-table and the tonsil dissected with the finger inserted into the mouth. Its inferior attachment, through which it receives its blood supply, should be severed with a cold-wire snare to prevent hemorrhage. This procedure is safe, leaves a clean wound, and assures singers, speakers, and others that future trouble from tonsillar disease need not be feared.

Faucial Mycosis—Mycosis Tonsillaris.

Etiology: This is a form of bacterial disease, or rather a form of bacterial growth, attending certain morbid states of the fauces, but more particularly the tonsils. The growth or fungus belongs to the schizomycites group. A number of kinds of leptothrix are found in the mouth and fauces. They are regarded as non-pathogenic. They are slender rods staining with iodine solutions, thereby showing they contain starch. They pile up on end, forming tufts of the prickly wart style. When seen in the acute stage they are of a soft creamy consistency, and may be wiped from the membrane easily. When they are of long standing they are firmly attached to the epithelial layer of the mucosa and are difficult of removal. Lacunar disease of the tonsils strongly favors their development. Live tissue has no attactions for them, while dead epithelial cells are their favorite tissue attachments. Keratosis of the tonsils is therefore a strong predisposing factor in their development. The author has often seen mycosis buccalis at the point where the upper and lower teeth come together, in other words, where keratosis occurs as a result of friction.

The size of the tufts varies from that of a pin-point to that of a small wart.

Faucial mycosis—treatment: Mouth washes and gargles of chlorate of potassium and other antiseptic solutions exert but slight influence on the growth. Equal parts of guaiacol in olive oil or sweet oil of almonds applied locally seems to be of decided benefit. Salicylic acid has its enthusiastic supporters, but the facts are that nothing short of the removal and cauterization of the points of attachment is of positive value in their complete eradication. When they are once thoroughly removed their return is still quite probable.

If the tonsils are enlarged their removal exerts a favorable influence on the growth.

Benign Tumors of the Fauces.

Varieties: On account of the character of the histological formation of the fauces benign neoplasms are not often found here.

Papilloma of the uvula, soft palate, or faucial pillars is the most common form of neoplasm seen in this region. These growths appear as warty vegetations and show little disposition to extend. Occasionally they grow quite rapidly. As they present the usual appearance of papillomata growing upon mucous membranes generally, an extended description of them will not be given here.

Papilloma of the uvula—treatment: This consists in removal with a snare, scissors, or knife. A portion of the healthy mucous membrane should also be removed and the point cauterized to prevent a return of the growth.

Fibroma of the soft palate: It develops rapidly and has no other symptoms than those due to mechanical interference. If operated upon early there seems to be no danger attending removal.

Angioma of the fauces: Several cases have been reported. It consists of a network of small bloodvessels with very thin walls. The surface of the tumor is quite sensitive and gives rise to considerable pain upon deglutition.

The *treatment* consists of radical removal with the cautery loop, as the hemorrhage would otherwise be severe.

Malignant Tumors of the Tonsils.

These are comparatively rare affections; 6 in 1000 of all diseases of the fauces and pharynx being malignant.

The **varieties** found may be thus classified:

a. Epitheliomata.
1. Squamous.
2. Alveolar.
3. Columnar.

b. Sarcomata.
1. Round-cell.
2. Spindle-cell.
3. Lymphosarcomata.
4. Angiosarcomata.

Occurrence: Round-cell sarcoma is the most common type of malignant disease of this region according to some authorities; while, according to others, epitheliomata are more common. Sarcoma of the tonsil seems to be a disease of adult life, whereas in other parts of the body it is peculiar to the earlier periods. Epitheliomata belong to the later periods of life. They are found most often in males, although the difference is not marked enough to make this distinction of practical importance.

Etiology: The location and function of the tonsils and fauces, and the irritation to which they are subjected in a measure account for the great frequency with which they are affected by malignant disease. The low resisting power of all lymphoid tissue must also be taken into account. The use of alcohol and tobacco seems slightly to predispose to malignant disease of the fauces. Heredity has the same influence as in tonsillar inflammations.

It was my privilege, quite recently, to examine a case of epithelioma under the care of Dr. Sherwood. The tumor was ulcerating beneath the angle of the jaw and within the pharynx.

The **pathology** of malignant tumors of the fauces and tonsils differs in no way from such morbid processes elsewhere in the body; hence will not be described in detail in this work.

Symptoms: The early symptoms of sarcoma and epithelioma are those due to mechanical interference; a little later there is the addition of sharp lancinating pain.

Prognosis: Sarcomata affecting the *mucosa* may not be a very grave affection. Under early operative interference a cure may be expected. When, however, it attacks the substance of the tonsil it becomes a grave disease, and the prognosis should be guarded. The tonsil being a part of the lymphatic system makes the disease much more liable to extend to contiguous parts.

Epithelioma of the tonsil or fauces is grave in the extreme. It is so located as to favor its rapid spread to adjacent lymphatic glands.

Malignant tumors of the tonsils—treatment: Operative measures offer the only hope. These can usually be done through the mouth; hence no special description of the primary incisions through the skin and underlying tissues will be given. In some cases a preliminary tracheotomy should be done. This question must be decided in each individual case according to the symptoms. The carotid artery may be ligated as a preliminary step; otherwise severe hemorrhage may occur. Should it be necessary to gain entrance into the pharynx through the lateral wall, the choice will lie between a lateral pharyngotomy and a subhyoidean pharyngotomy.

Operative interference in epithelioma only offers the hope of postponing a fatal issue a few months at the most.

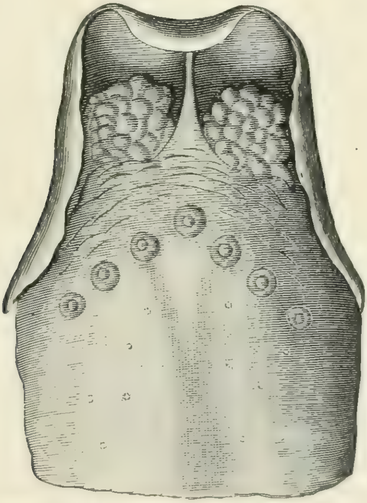
THE LINGUAL TONSILS.

Anatomy: These are composed of masses of lymphoid tissue in the glosso-epiglottic space; or, as is commonly said, they are on the base or root of the tongue. They are slightly elevated nodules, some of which are pitted or concave on account of shallow follicles. There are present numerous mucous and seruminous glands, which are not found in "tonsils" elsewhere. The veins at the base of the tongue are very superficial, and might be mistaken for enlarged lingual tonsils.

Inflammations of the Lingual Tonsils.

Varieties: The intimate connection between the lingual and the other tonsils causes them to respond to the same influences. Inflammation at the base of the tongue is rarely primary, but usually attends inflammation of the higher tonsils. The same kinds of morbid processes occur in the lingual tonsils, namely, acute, subacute, and chronic inflammations. The crypts become filled with epithelial cells, leucocytes, and fibrinous exudate. They undergo hypertrophy as do their larger neighbors above. Deep and superficial abscesses occasionally form here also.

FIG. 122.



Hypertrophy of the lingual tonsils. (Cohen.)

Lingual tonsils—symptoms of inflammations: A sense of fulness and slight pain upon deglutition and vocalization are the chief symptoms. Upon inspection with a laryngeal mirror the lymphoid nodules will be found to be enlarged and red; and if the crypts are involved a yellowish material may be seen in them. Should there be an abscess of this region the symptoms become quite marked; the pain is intense, the fever high, and deglutition of fluids difficult or impossible. Dyspnœa may develop from obstruction to the glottic respiratory tract. The abscess may perforate the lingualis muscle and become or simulate a true *angina Ludovici.*

Prognosis: *Catarrhal* and *lacunar* inflammations run their course within a few days or weeks; while the suppurative form may extend and give rise to serious symptoms. Œdema of the glottis is rare. Should dyspnœa of a dangerous nature occur tracheotomy should be performed.

Inflammation of the lingual tonsils—treatment: This should be much the same as for inflammation of the faucial tonsils. On account of the depression resulting from the dyspnœa and difficult deglutition stimulants are especially indicated in the more acute or phlegmonous types. If an abscess is present, it should be opened with a curved tonsil bistoury. In making the incision care should be taken to avoid the lingual arteries.

Hypertrophy and Varix of the Lingual Tonsils.

The same **causes** that have already been given in reference to disease of the lymphatic tissue of the faucial tonsils hold true with the lingual tonsils. Oft-repeated inflammations of whatever source cause hypertrophy and varix of the lingual veins. The two conditions are usually associated; hence they will be considered together.

Hypertrophy and Varix of the Lingual Tonsils—Symptoms.

The **subjective symptoms** may be classified as follows:

1. A feeling of fulness and irritation due to mechanical interference with the normal functions and movements of the parts. There is a more or less constant association of hypertrophy and varix at the base of the tongue with the condition known as *globus hystericus.* This seems to point to this condition as a physical explanation of what has heretofore been

thought to be purely a functional nervous phenomenon. Patients describe the sensation in the glosso-epiglottic space as seeming to be caused by the presence of a bead, pea, straw, blister, or other foreign body. These signs disappear during deglutition, but quickly return.

2. Torticollis has been noted as an occasional sign.

3. The singing voice is impaired, not so much by the direct pathologic interference as by the inability properly to vocalize. Continued attempts to do so cause the voice to become impaired. In other words, there would be no voice symptoms should the singer discontinue his special efforts in that direction.

4. Cough and hemming are prominent subjective symptoms. The effort is ineffective; the cough is of a brassy, croupy character.

5. Paroxysmal dyspnœa is an occasional symptom.

The **objective** or **physical signs** as revealed by the laryngoscopic mirror are:

1. A unilateral or bilateral mulberry-like mass (Fig. 122), which more or less obscures the epiglottis. In some cases the hypertrophy not being so marked, the tonsils appear as several small discrete nodules with varicose veins running between them.

2. The epiglottis is crowded backward, thereby obscuring the view of the larynx.

3. The mucosa of the larynx is usually hyperæmic, while the cords are nearly normal.

4. Passive hemorrhages are often observed. The blood which has accumulated during the night is expectorated in the morning. This doubtless occurs during the daytime also; but as it is very slight, and the acts of swallowing and expectoration occur at frequent intervals, it is not noticed.

5. Faucial tenesmus, laryngeal spasm, and asthma have been reported as occasional symptoms of hypertrophy of the lingual tonsils.

Hypertrophy and Varix of the Lingual Tonsils—Treatment.

Treatment offers a good prognosis. It should be both general and local.

The **general or constitutional treatment** should be addressed to the strumous or other diathesis which may be active in the individual case. Exercise in the open air and sunshine is of more importance than the administration of drugs. This being a disease of adult life, the abuse of alcohol and tobacco may be exciting causes, hence their discontinuance in such cases should be advised.

The **local treatment** is palliative and surgical or semisurgical in character. The irritations, foul secretions, etc., which attend this and the other faucial, pharyngeal, and nasopharyngeal disease should be properly treated with antiseptic and emollient washes. The mineral astringents reduce the congestion, which is a prominent feature in the earlier stages of the disease. Lugol's solution :

℞ Potass. iod.,	gr. xlviij ;
Aquæ dest.	℥j.—M.

Sig. Apply with a cotton-mop to the lingual tosnils.

This is a favorite treatment in Europe.

The sulphate of zinc, tannin, perchloride of iron or aluminum are also useful in relieving congestion. Where there is pain or distress a 50 per cent. solution of guaiacol in olive oil affords relief.

Caustic applications, as lunar caustic, chromic acid, and glacial acetic acid, are inefficient and dangerous remedies. They may gain entrance into the stomach and produce serious consequences.

The **galvanocautery** is a good and safe method of removing the lymphoid masses or for destroying the varix. (1) The parts should be cocainized with a 10 to 20 per cent. solution. (2) The patient should then protrude his tongue and grasp it with a napkin, as in laryngoscopy. (3) The operator introduces the laryngeal mirror with the left hand, while (4) the cautery electrode is brought to bear upon the lingual tonsil, or varicose veins. (5) The electric current should be connected and the (6) parts seared with the red-hot platinum point. If the veins only are to be treated, a small point should be used ; while a flat electrode is more desirable for the treatment of the tonsils.

The **cold- or hot-wire snare** are excellent methods of operating. The cold wire used with the Bosworth snare is probably the better and safer of the two. If the tonsil is broad at its base, it may be necessary to transfix it with a needle and then pass the wire around it.

Specially devised tonsillotomes conforming to the curve of the dorsum of the tongue are now made by instrument-dealers, and the operation is thereby simplified. They act on the principle of the Mackenzie or Mathieu tonsillotome. One caution only will be given, namely, use the laryngoscope so as to avoid clipping the top of the epiglottis.

The operation is followed by complete relief from the distressing symptoms, but there will be some local pain and soreness for a few days.

The attendant disorders of the fauces, nose, and nasopharynx should also receive appropriate surgical treatment.

Lingual Tonsils—Other Diseases.

The **exanthems**, as scarlet fever; **specific inflammations**, as diphtheria, syphilis, lupus, tuberculosis, and leprosy; **neoplasms**, as fibroma, lymphoma, and malignant tumors, may affect the lingual tonsils.

POSTNASAL ADENOIDS.

Synonyms and definition: Hypertrophy of the Pharyngeal Tonsil; Adenoids; Postnasal Vegetations. These and other names have been used to designate enlargement of the normal lymphoid masses on the vault and posterior superior wall of the nasopharynx (Fig. 123).

Postnasal Adenoids—Etiology.

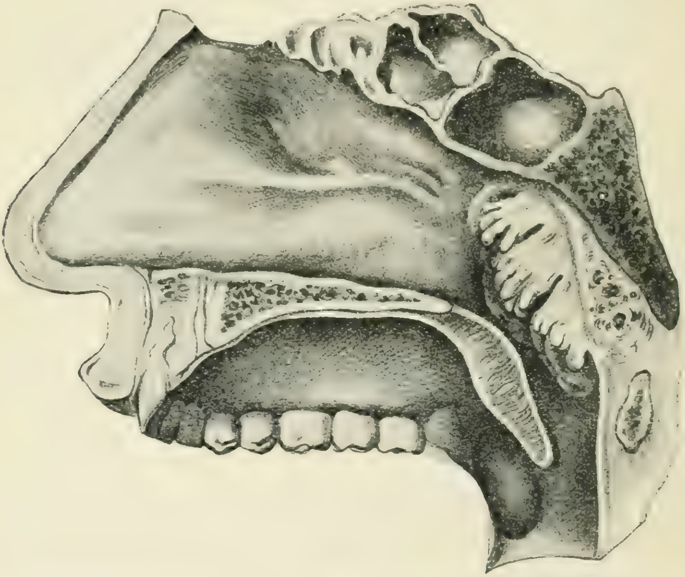
Age is of prime importance in the causation of hypertrophy of the pharyngeal tonsil. Most cases develop before the age of puberty, at which time they usually begin to atrophy.

It may be a congenital condition. The author has seen cases which were unable to nurse on account of the nasal stenosis; and which after the removal of the adenoids were able to do so.

Cases are reported in persons more than seventy years old. They are rare, however, in advanced years, and when present may be of rather recent growth from inflammatory causes.

In most persons affected in youth only a trace is left after the thirtieth year. Just prior to puberty a retrograde metamorphosis in the form of colloid degeneration usually affects the pharyngeal tonsil.

FIG. 123.



Anteroposterior section of the head of an adult, showing the situation and gross structure of hypertrophy of the lymphoid tissue of the nasopharynx. (Zuckerkandl.)

Sex does not appear to be a decisive factor in the production of this disease. The experience of various observers, while they differ somewhat, yet when taken as a whole, show more cases in the male. Theoretically boys should be more subject to it than girls, because they are more exposed to the inclemencies of the weather. On the other hand, girls are confined indoors more than boys, and are in consequence more tender and subject to slight irritations.

Either **constitutional conditions** are a cause of postnasal adenoids or the adenoids produce the constitutional conditions. They are usually associated; writers differing as to which is cause and which is effect. Most authorities, however, agree that constitutional conditions are potent predisposing causes. They are found associated with rickets, infantile convulsions, and laryngismus, as well as with other strumous manifestations. Tuberculosis has been assigned as an important etiologic factor. The tubercle bacillus has been searched for in the tissue, cultures grown from macerated tonsils, etc., in the endeavor to establish the tuberculous diathesis. Thus far the investigations have been open to criticism. My own experience leads me to doubt a constant relationship. The lymphatic temperament has been assigned as a cause. Adenoids are often found in deaf-mutes.

Heredity perhaps has little influence in the development of postnasal adenoids. This may seem contradictory when it is stated that the growths often appear in several members of the same family. The explanation of this apparent contradiction is in the fact that certain anatomic peculiarities are hereditary, and it is these peculiarities that cause or aid in causing the adenoid growths. Under *Anatomic Considerations* (next page) these peculiarities will be further considered.

Race is a determining factor, as the following citations will show: Adenoids are common in Australia, Queensland, China, Greenland, extreme North America, Northern Europe, Argentine Republic, and the United States. They are comparatively rare in Sumatra, Siam, and Spain. The Hebrew race are particularly prone to be thus affected.

Climate perhaps in a measure accounts somewhat for the above distribution. It is certain that humid cold climates favor adenoid growths in children, while warm, humid climates minimize the hypertrophic tendency.

Inflammatory action in the nose and nasopharynx, as seen during an attack of the exanthems (specific fevers), plays an important *rôle*. Diphtheria, scarlet fever, and influenza are perhaps the most active of the inflammatory irritative agents. Common colds are also somewhat responsible for exciting lymphoid hypertrophy in the nasopharynx.

Purulent nasal catarrh may be a cause as well as an effect. It is a known pathologic law that lymphoid tissues in children have a tendency to enlarge under **slight irritations.**

Anatomic Considerations.

Having reviewed most of the various etiologic factors at work in the production of postnasal adenoids, we will now discuss what is the most important of them all, namely, **nasal stenosis.** It has been well proven that transient but recurrent anterior nasal stenosis is attended by rarefaction of the air in the nasal fossæ posterior to the obstruction during inspiration. The rarefaction causes hyperæmia of the mucosa (especially the erectile tissue) of the nose and nasopharynx. Chronic hyperæmia may ultimately follow and be attended by hypernutrition; hence hypertrophy of the parts. The lymphoid structures in the vault of the pharynx, being especially susceptible to irritation, rapidly grow to be of large size.

Some have regarded a **high-arched palate** as a cause of nasal stenosis and postnasal adenoids. The height of the arch is usually more apparent than real. The superior maxillæ in such cases are contracted and the teeth longer, on account of the mouth being so constantly open, pressure thereby being removed.

Cleft palate is another anatomic source of irritation to the nasopharyngeal space.

It should be remarked that *constitutional taints* may remotely influence any or all of the foregoing causes. In other words, it is still a mooted question as to whether there is not a **constitutional** struma or **dyscrasia** which will not only account for postnasal adenoids, but for the so-called causes as well.

Postnasal Adenoids—Histopathology.

There are **normally** groups of lymphoid tissue or glands in the vault and posterior wall of the nasopharynx. They extend from the *bursa pharyngea* in the median line across the upper and back part of the pharynx. The tissue is of a low embryonic type, and is excited to hypertrophy under slight irritation during childhood. The nodules are of irregular shape and distribution. The crypts of the pharyngeal, unlike those of the faucial and lingual, tonsils are absent or ill developed. The surface is covered by a single layer of ciliated columnar epithelium, except where contact and movement against opposing structures have caused their disappearance. Mingled with the ciliated epithelium are numerous chalice-cells, which in a measure account for the abundant thick mucous secretion in the nasopharynx. There are no true mucous glands, but there is a single row of lymph-nodules near the surface. In the deeper layers there is a heavy reticulum, the meshes of which are filled with small lymphoid cells each containing one or two nuclei.

The gross **morbid appearance** of the growth as seen *in situ* is usually a pearly-gray, cushion-like mass, slightly nodular. As has been aptly expressed by Meyer, when the finger is introduced into the nasopharynx this mass gives a sensation like "pushing the finger into a bunch of earthworms." More rarely the growth is very nodular, or even fimbriated, as is shown in most drawings in text-books.

Postnasal Adenoids—Symptoms.

The **physiognomy** affords the most striking and at the same time characteristic symptom of the disease. The mouth is open, the nasolabial furrow absent or slightly developed, the bridge of the nose widened, the cheeks flattened, the inner canthi drawn down, the root of the nose puffy and œdematous, and the eyelids slightly drooping. Taken *en semble*, the face has a stupid, sad expression which cannot but attract attention and sympathy. The alæ nasi are depressed at the angle between the superior and inferior lateral cartilages. No other form of nasal stenosis gives rise to this symptom.

The **objective signs** in addition to those given under physiognomy are those to be seen or felt in the pharynx, nasopharynx, fauces, nose, and chest. The pharynx usually presents a granular or nodular surface covered with frothy mucus. The lymph-nodules are those involved in follicular pharyngitis elsewhere described. They are somewhat œdematous and succulent in appearance. Occasionally there is pharyngitis sicca, which is not attended by rhinitis sicca. This seems strange at first thought, as the condition is usually secondary to rhinitis sicca. The explanation is to be found in the adenoid growth closing the superior and middle meatuses of the nose, thereby compelling the inspired air to traverse the inferior meatus, wherein it is not sufficiently humidified. It passes into the pharynx too dry and abstracts moisture therefrom, irritation results, and pharyngitis sicca is established. The uvula is œdematous and occasionally drawn to one side on account of paresis of the muscles on the opposite side. The pillars and velum are congested and of a dusky-red color. Pigeon-breast is an associated condition which has been variously explained. Coolidge reports six cases of postnasal adenoids or mouth-breathers, in which there was malformation of the lower extremities.

Aprosexia, or *mental dulness*, is in most cases more or less present. Various theories have been advanced in explanation of it. Axel Key and Retzius demonstrated the existence of lymphatic communication between the meninges and the nose and vault of the pharynx, and attribute the mental hebetude to obstructions of these channels.

Sleep is disturbed, the child tosses in bed, throwing off the covers, and usually snores on account of the mouth-breathing and paretic condition of the velum ; each inspiration causes the relaxed membrane to vibrate. The mental state during sleep is disturbed by dreams and nightmare. Upon awakening, these mental impressions often persist for some minutes, giving rise to what has been described as slight delirium. The snoring sometimes terminates in laryngeal stridor, and even in mild general convulsions. Upon awakening, the mouth and throat are dry. General malaise is often experienced in the morning, but disappears as the day advances.

Reflex phenomena often attend, if they do not result from, the adenoid growths. Hay-fever, paroxysmal sneezing, asthma, chorea, epilepsy, torticollis, headache, enuresis, genital irritation, exaggerated peristalsis of the intestines resulting in prolapse of the rectum have been reported as concomitant symptoms of the presence of postnasal adenoids.

Hearing: Disturbances of the function of hearing is one of the most common attendant symptoms of adenoids. Fully 60 per cent. of all persons with adenoids in the pharynx have more or less interference with audition. As small children often have earache and recover from it, the family and family physician have grown to look upon all ear affections in children as a necessary evil which time alone will heal. This view is responsible for innumerable defective ears in later life, and is often the indirect cause of death from meningitis, pneumonia, etc. This the author shows in the chapter devoted to Chronic Suppurative Otitis Media.

The *pathologic changes giving rise to ear symptoms* are (*a*) hyperæmia and catarrh of the mucosa of the Eustachian tube; (*b*) hyperæmia and catarrh of the tympanic cavity; (*c*) suppurative inflammation of the tympanic cavity; (*d*) the adenoids may impinge upon the orifices of the tubes and cause closure thereof; (*e*) retraction of the manubrium mallei and drumhead. The last-named condition when present in a child is almost pathognomonic of postnasal adenoids. The manubrium is very much retracted, hence foreshortened; the "cone of light" either broken or displaced; and the processus brevis very prominent.

A Physiologic Statement Explanatory of Some of the Preceding Symptoms.

The term "**mouth-breathing**" implies the absence of "nasal respiration." In other words, the respiratory functions of the nose are lost to the physical economy.

The **respiratory functions** are: (*a*) to warm or cool; (*b*) to moisten; and (*c*) to filter the inspired air. McDonald has clearly demonstrated that air after passing through the nasal chambers is either lowered or raised almost to the body-temperature. The bronchi are supplied with but few glands and secrete little moisture. The erectile tissue and serous glands of the turbinated bodies throw out about sixteen ounces of serum in twenty-four hours. The warmed and expanded air in the nasal chambers absorbs the greater portion of it, and it is thus carried to the lower air-tract, where it is needed for physiologic purposes.

When the temperature of the air is elevated its capacity to absorb moisture is greatly increased. This has an important bearing upon the question under consideration. The air is elevated in temperature in its passage through the nose, pharynx, and bronchial tubes; but failing to receive its full capacity of moisture in the nose, it abstracts it from the air-tract below. Some have held that the moisture taken up in the nose is to be deposited in the bronchi and air-cells of the lungs, which are but sparsely supplied with mucous and serous glands. This is questionable if taken as a statement of the entire truth. Undoubtedly this is one of the purposes. Another purpose is so to charge the inspired air with moisture that when it reaches the lower air-tract it will not absorb the moisture secreted there for physiologic purposes.

The **nasal chambers** are so constructed that the column of inspired air comes in contact with the moist Schneiderian membrane where the dust, germs, etc., are deposited. The air is thereby filtered, and enters the lower air-tract free from obnoxious foreign particles.

As **a consequence of the impairment or loss of nasal respiration**, the inspired air passes into the lower air-tract insufficiently tempered, insufficiently moistened, and insufficiently filtered. The mouth and pharynx but feebly perform the respiratory functions of the nose. The mucous membranes of the larynx, trachea, and bronchi are irritated and manifest a catarrhal tendency. The epithelial cells lining the air-vesicles (of which there is but a single layer) become irritated also. The cells at first swell, and finally pile up until the lining is two, four, six, ten, or even twelve layers deep in places. I have demonstrated the foregoing statement by microscopic examination of the air-vesicles of guinea-pigs that had been submitted to prolonged exposure to dry dust-laden air.

Thickened epithelial lining of air-vesicles: Because of this thickening a deficient amount of oxygen is absorbed by the blood. Faulty oxygenation of the tissues or faulty metabolism results. The half-way products of oxygenation, as uric acid, etc., are freely formed and circulate through the system. Another result of the thickened vesicle-walls is the retardation of the elimination of carbonic-acid gas. In other words, carbon dioxide is accumulated in excess in the blood. When it is in excess it acts as a violent poison to the leucocytes. The function of the leucocytes is thereby impaired.

Function of the leucocytes impaired: We as yet know but little of the part played by the leucocytes, but we know enough to apply the facts to the purposes of this essay. We may name germicidal and scavengerial properties as among the fairly well established functions of these corpuscles.

Physical malformations and nervous phenomena: If we but pause, and recall the train of results following chronic irritation of the epithelial lining of the air-vesicles, it becomes apparent that physical malformations and nervous symptoms must of necessity largely make up the clinical picture of mouth-breathing. Deficient oxygenation of red blood-corpuscles leads to imperfect metabolism. Excess of carbon dioxide leads to impairment of the function of the white blood-corpuscles. Malnutrition results in physical imperfection. The scavengerial function of the leucocytes being impaired allows the accumulation of irritants in the blood, which in their turn cause peevishness, inattention, restless nights, and quick temper. The impairment of the leucocytal function renders the patient an easy prey to microbic infection, and it augments the nervous symptoms just enumerated.

Recapitulation: 1. Respiratory functions of the nose are to warm or cool, moisten and filter the inspired air.

2. In mouth-breathers these functions are absent.

3. The lower air-tract is unable to supply or is deprived of moisture.

4. An irritation of the epithelial lining of the air-vesicles results.

5. This irritation causes thickening of the vesicle-walls.

6. Deficient oxygenation of the blood results in:

7. Imperfect oxygenation of the tissues, half-oxidized products being thrown into the circulation.

8. The "half-way" products cause nervous phenomena.

9. Malnutrition leads to physical imperfection.

10. Excessive accumulation of carbon dioxide in the blood impairs: the functions of the leucocytes and other cellular structures.

11. Microbic infection is rendered easy, and the partially-

oxidized products are not removed from the circulation by the leucocytes. The nervous phenomena are thereby augmented.

The foregoing train of occurrences may be observed in children who are mouth-breathers, whether it be due to postnasal adenoids or to other forms of nasal obstruction.

Postnasal Adenoids—Treatment.

This is medical, surgical, and after-treatment.

The **medical treatment** is of but little value except as it affords temporary relief of some of the distressing symptoms. Alkaline and aromatic washes and sprays to the nose and nasopharynx remove accumulated secretions and diminish the congestion. Urban Pritchard recommends the following astringent application as of more value than other local remedial agents:

℞. Glycerit. acid. tannici, ʒiij;
Aq., ad ℥iij.—M.
Ft. inject.

Sig. One teaspoonful in a wineglass of water, to be injected *down* the nose, through the inferior meatus, night and morning.

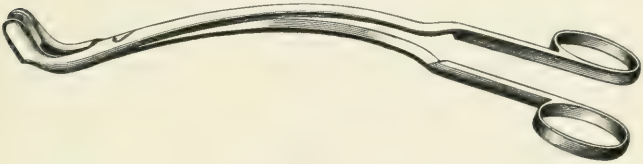
Alkaline washes and the Pritchard treatment may be pursued to relieve certain distressing conditions while waiting for the operative treatment.

Surgical measures are the only ones that are of permanent value. Various procedures, as scarification, superficial scraping, etc., to excite bleeding have been tried with the hope of affording permanent relief. Inasmuch as the condition is one of hypertrophy and not of congestion, such measures are inadequate. The *removal of the growth* is essential to complete success.

The **chief indications** for the removal of the adenoids are: "(1) well-marked signs of nasal obstruction, such as mouth-breathing, adenoid expression of countenance, alteration of voice; (2) deafness, or oft-recurring attacks of deafness, the result of middle-ear disease; (3) continually recurring head-colds, or nasal, laryngeal, or bronchial catarrh; (4) paroxysmal attacks of sneezing, hay fever, laryngismus, or spasmodic asthma; (5) defective physical and mental development, chronic tuberculous enlargement of the cervical glands."[1]

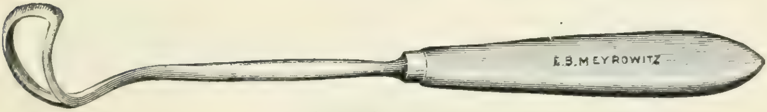
Various instruments have been devised for the performance of this operation, among which are the artificial nail or, as it is sometimes called, the thimble scraper; Meyer's ring-knife to be used through the nose; Loewenberg's forceps (Fig. 124) curved so as to be used through the mouth and introduced into the nasopharynx; the Gottstein curette (Fig. 125) or curved ring-knife to be introduced into the nasopharynx through the mouth; the galvanocautery and chemical caustics, only mentioned as dangerous and obsolete methods; the cold-wire snare may be used as suggested by Bosworth, but its use is somewhat difficult and should only be attempted in the absence of other suitable instruments. The finger-nail may be used in young subjects in whom the growth is still soft and yielding.

FIG. 124.



Loewenberg forceps.

FIG. 125.



Gottstein curette.

The **anæsthetic** may be local or general. Cocaine and eucaine solutions may be applied *locally* through the nostrils or through the mouth by means of curved cotton carriers saturated with the solution. Anæsthesia more or less complete may be thus produced. According to my own experience, it is not a very satisfactory method.

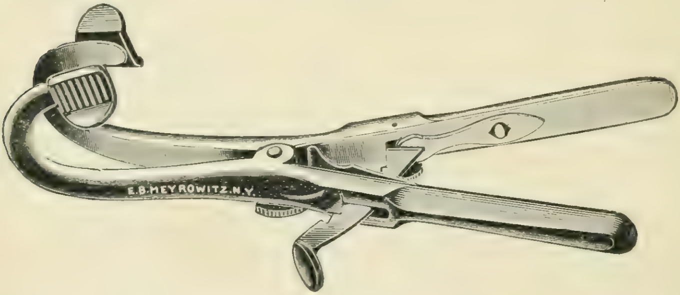
---

[1] Washham, Nasal Obstruction.

General anæsthesia is preferable and may be induced by the use of any of the recognized fluids and gases used by general surgeons. It is never necessary to carry anæsthesia so far as to abolish laryngeal reflexes. The pin-point pupil should be maintained. Chloroform seems to act unfavorably in children with adenoids and is to be condemned. If continental or English methods of procedure are to be followed, ether is perhaps the best. If the more rapid and equally thorough American method is adopted, laughing-gas (nitrous-oxide gas) or the bromide of ethyl is to be preferred as a simpler and safer anæsthetic. In my private practice I use the bromide of ethyl to the exclusion of all other anæsthetics.

The **instruments of most value** are: the Gottstein curette and Loewenberg forceps; a mouth-gag of cork or one of the many patterns (Fig. 126) to be purchased at instrument stores; and lastly the finger-nail of the right index-finger. A tongue depressor should be used in introducing the Gottstein curette and Loewenberg forceps.

FIG. 126.



Mouth-gag.

The **position of the patient** is a consideration of importance. If he is very young, I have an assistant take him on his lap and cross his legs over those of the child. The folded arms are held with his left hand and arm, while the head of the child is held against the right shoulder of the assistant with his right hand grasping the forehead. If the child is older and tractable, I have him sit in a high-backed chair, telling him to grasp firmly the edge of the seat. An assistant stands back of the chair, grasping the patient's head between his hands. The anæsthetic is administered while in this position.

The **operation is performed** as follows: The assistant back of the chair introduces the mouth-gag on the patient's left side. The operator depresses the tongue and with the tonsillitome removes first the right faucial tonsil, reverses the instrument (Mathieu's), and removes the left. The throat is rapidly sponged free of blood and the Gottstein curette is introduced through the mouth into the nasopharynx, care being taken to carry it well up into the vault, and to the left side (Figs. 127 and 128). The handle is now made to describe the arc of a large circle, the fulcrum being the top of the fauces. The instrument is again carried to the vault in the median line and the same movement of the handle repeated. A third time the instrument is carried to the vault, but on the right side, and the same procedure repeated. The instrument should be pressed firmly and evenly to the vault and posterior wall during the upward movement of the handle (Fig. 129) of the curette. Three or four sweeps of the curette are usually quite sufficient to remove most of the growth.

FIG. 127.

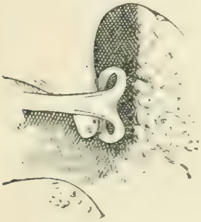


FIG. 128.

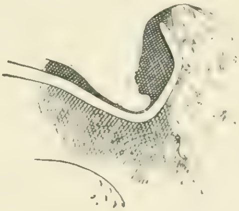
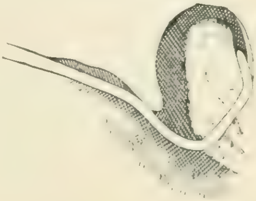


FIG. 129.



After the *removal of the instrument* the throat is again sponged free of blood and if necessary the head and body inclined forward to prevent blood entering the trachea. The operator now introduces the index-finger of the right hand into the nasopharynx, therewith locating and removing any fragments remaining. It is needless to remark that the finger and nail should be made surgically clean before the operation. The time required for the whole procedure need not be more than two to five minutes.

If the child is placed upon an operating-table, his head should hang back and down over the end of it, or he should be placed upon his left side. It is much more awkward for the operator in either of these positions and a longer time will be required to perform the operation.

After-treatment: The patient's head and body should be kept well forward and down until he regains consciousness to prevent the blood entering the œsophagus and trachea. When he regains consciousness he should be directed to blow his nose in order to remove blood clots or portions of the adenoid growth that may have lodged there.

The nose and pharynx should not be syringed for twenty-four hours after the operation if there is danger of exciting otitis media by so doing. An antiseptic or mildly astringent gargle may be used to relieve the fauces and pharynx of the thick secretion during the healing process.

As considerable quantities of blood are apt to be swallowed during the operation, it is advisable to administer a cathartic on the evening of the day of operation.

Complications attending or following operation: 1. There is danger of suffocation from the presence of blood in the trachea.

2. There may be delirium or bewilderment following the operation.

3. If the instrument is carried too far to the side of the pharynx, the orifice of the Eustachian tube may be injured, and a subsequent stricture result.

4. Secondary hemorrhage is rare.

5. The operation may be followed by infection of the middle ear and mastoid, and of the cellular tissue of the pharynx. A true phlegmonous pharyngitis or abscess may occur.

6. Latent malaria has been converted into the active type immediately after the operation.

7. Dangerous hemorrhage may occur as a result of excessive fibrosis; or of the abnormal placement of the internal carotid and vertebral arteries. When dangerous hemorrhage takes place it may be controlled by digital pressure; by pledgets of cotton saturated with a 10 per cent. solution of antipyrin; by eating crushed ice; by injections of hot water, etc.

THE LARYNX.

EXAMINATION OF THE LARYNX.

Good illumination and the proper instruments are essential to the examination of the larynx.

The **means of illumination** will of necessity depend very much upon the environment of the physician. If, for example, he is located in the country, where gas and electricity are not to be had, he must depend upon sunlight or the oil-lamp.

Sunlight furnishes an ideal illumination, but for obvious reasons is not always available. An ordinary *oil-lamp* is always available in most country places and is quite satisfactory.

In the larger cities, however, where gas and electricity are in almost every home and office building, their use is more practical, and with the proper appliances furnish a more brilliant illumination.

The ordinary flat gas-flame, the argand burner, or the hooded Welsbach gas-burner may be used. The Welsbach burner makes an ideal light, as it is both brilliant and steady. *Gasoline* is now utilized, by means of a very simple contrivance, to furnish gas for the Welsbach burner, thus making it possible for the practitioner in the most remote places to have an ideal mode of illumination.

Electricity is, however, the light *par excellence* for the specialist, as it is cleanly, and throws out less heat, which is a desideratum of no small importance where the lamp or gas-light may be in constant use for hours at a time.

Instruments: If sunlight, the oil-lamp, gas, or the electric incandescent globe are utilized for the source of illumination it is necessary to use a concave head-mirror to throw the rays of light into the oral cavity. There are electric head-lamps which do away with the necessity of using a mirror reflector. The Phillips and the Kirstein head-lamp are perhaps the best on the market. The Kirstein is better adapted for ear work as it throws the rays of light in a direct line with that of vision.

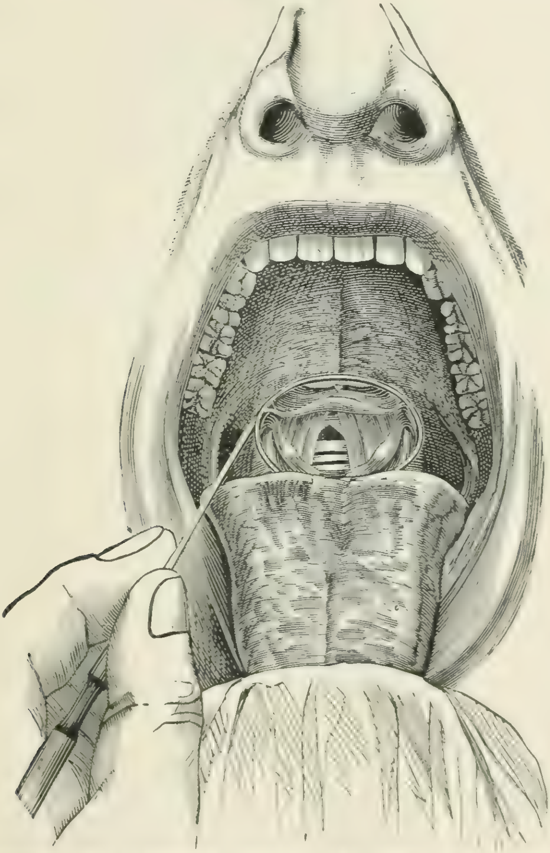
In addition, a napkin for holding the tongue, a tongue depressor, and three or four laryngeal mirrors of various sizes should be at hand for making the examination. The larger sized ones are generally preferable for getting the laryngoscopic image.

Method of examination: It is the author's custom to proceed somewhat as foliows: The patient is placed on a revolving chair a little to the front and left side of the light, and at such a height that his mouth is about on a level with my chin. He is instructed as to what I intend doing, and as to how he can aid me in doing it. The mental conception of what I expect of him enables him to adjust the tongue, fauces, and epiglottis so as to permit of an easy examination of the larynx.

After having adjusted the light about nine inches to the right and a little back of the patient's ear, the patient is told to protrude his tongue, the end of which I seize gently, but firmly, between the folds of a napkin (Fig. 130), and pull gently forward and downward, using the index-finger beneath the tongue as a fulcrum. This simple precaution avoids giving the patient pain by dragging the tongue down over the teeth. I direct him to relax the lower jaw and tongue, and breathe with quick, short breaths. While he is in the act of doing this, I insert a large warm laryngeal mirror, holding it as a pen, keeping the back of it well up toward the roof of the mouth, following the curve of the vault back to the uvula and soft palate, which I lift up gently, but firmly, if need be, against the upper and posterior wall of the pharynx. Especial care should be exerted to avoid touching the tongue upon its posterior aspect, as violent retching may be thus produced.

The face of the mirror should now look forward and downward, and the light from the head-mirror or lamp be directed upon it. The rays will thus be reflected downward upon the larynx, and the image returned to the eye. The examination should be done quickly and skilfully, as the parts will not long tolerate the presence of the mirror. Should retching or coughing be excited the examination should be discontinued at once,

Fig. 130.

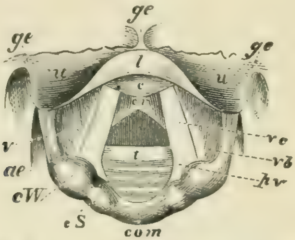


Laryngeal mirror in position, displaying the laryngeal image. (Cohen.)

and the patient's mind diverted from his throat. A few sips of ice-water will usually allay the irritability of the parts, and the examination may be repeated.

The position and action of the vocal cords and arytenoid cartilages may be brought into view by directing the patient to utter the syllables "eh," "ee," and to take a long deep breath.

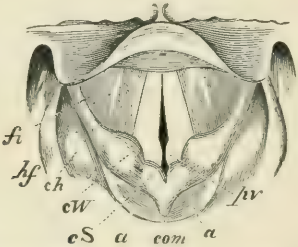
Fig. 131.



Laryngoscopic diagram, showing the vocal cords widely drawn apart, and the position of the various parts above and below the glottis, during quiet breathing (from Mackenzie): *ge*, glosso-epiglottic folds; *u*, upper surface of epiglottis; *l*, lip or arch of epiglottis; *c*, protuberance of epiglottis; *v*, ventricle of the larynx; *ae*, ary-epiglottic fold; *cW*, cartilage of Wrisberg; *cS*, cartilage of Santorini; *com*, arytenoid commissure; *vc*, vocal cord; *vb*, ventricular band; *pv*, processus vocalis; *cr*, cricoid cartilage; *t*, rings of trachea. (Seiler.)

The laryngoscopic image: The image is an inverted one, as it is only the reflection from the mirror that strikes the retina (Figs. 130–132). To remember this is of importance in making topical applications and in operating upon the larynx. The movements of the instruments thus used will be the

Fig. 132.



Laryngoscopic diagram, showing the approximation of the vocal cords and arytenoid cartilages, and the position of the various parts, during vocalization (from Mackenzie): *fi*, fossa innominata; *hf*, hyoid fossa; *ch*, cornu of hyoid bone; *cW*, cartilage of Wrisberg; *cS*, cartilage of Santorini; *a*, arytenoid cartilages; *com*, arytenoid commissure; *pv*, processus vocalis and cartilages of Seiler. (Seiler.)

reverse of that which seems natural, and considerable experience will be required to accustom the operator to them. The beginner should provide himself with a model mouth and larynx, or construct one of a piece of large tubing, and practice upon it with the laryngeal mirror, reflected light, and curved laryngeal instruments until he is accustomed to the reverse movements reflected from the laryngeal mirror.

Autoscopy or direct laryngoscopy : Kirstein is the author of this method of examination, which, while it is not universally practicable, is, nevertheless, a very valuable addition to our means of diagnosis and operation. It is rather difficult to do, although it seems quite simple. The obstacles interfering with its use may be overcome by a careful study of the technique of the procedure and by experience in its application.

The only *instrument* required is a specially designed tongue depressor, which has a long spatula, slightly curved downward at its end so as to conform to the curve of the base of the tongue. The curved end is thick and rounded, so as to avoid injury to the tongue.

The *method* of using it is as follows : The patient should be seated in a chair, leaning slightly forward with head inclining upward, so as to bring the oral in direct line with the pharyngeal and laryngeal cavities. The tongue depressor is then introduced as far back as possible on the tongue, and firmly pressed downward and forward so as to overcome the arch of the tongue and thereby bring the larynx into plain view. Special care should be taken to avoid using the upper teeth as a fulcrum. The operator, who is standing in front of the patient, can, by means of the Kirstein head-lamp, see the larynx in its true position.

By this method the larynx can be operated upon with straight instruments instead of the usual curved ones. This method, even in the hands of an experienced operator, is apt to occasion considerable discomfort to the patient unless he is under the influence of a general anæsthetic.

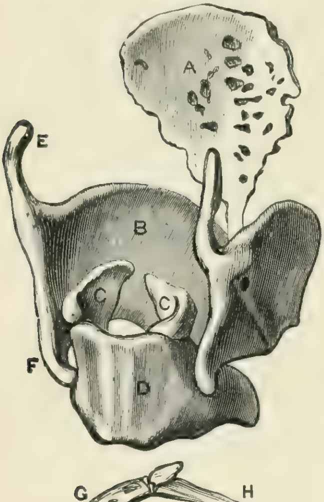
Laryngoscopic Landmarks.

The general anatomy of the cartilages and muscles is shown in Figs. 133–135.

The laryngoscopic mirror will show the following landmarks (Figs. 131 and 132):

The **epiglottis** with its crescentic or V-shaped edge standing up against the base of the tongue. It occupies the upper portion of the mirror and presents its concave surface downward.

FIG. 133.



Hyoid bone and the laryngeal cartilages: G, body of the hyoid bone; H, large cornu; J, small cornu; A, epiglottis; B, thyroid cartilage; C, arytenoid cartilage; D, cricoid cartilage; E, upper cornu; F, lower cornu of of the thyroid cartilage. (Ellis.)

It should be *examined* for deformities, neoplasms, catarrhal inflammation, and tubercular, syphilitic, and malignant infiltrations. Its color should be noted as should also the condition of the veins and arteries upon its surface.

The next landmarks to engage the attention are the *rounded prominences* in the lower field of the mirror. They are covered with mucous membrane and mark the location of the **arytenoid cartilages.** They play a prominent part in vocalization and are often the seat of tubercular infiltration. Their color, size, and symmetry should be noted. If the mucosa covering them is pale and ash-colored there is a strong probability that the patient is affected with tuberculosis. At a later stage they may be much enlarged from tubercular infiltration or broken down by ulceration. One side alone may be thus affected.

The **vocal cords** appear as pale, pinkish bands, occupying the central portion of the mirror in its perpendicular axis. They seem to unite in the upper portion of the mirror and to spread apart as they pass into the lower field. A reversal of the image gives the true position of the parts, namely: the cords unite anteriorly at the base of the epiglottis, and pass backward to the arytenoid cartilages. During deep inspiration the cartilages separate, and thus cause the cords to spread apart in the lower field of the mirror.

Fig. 134.

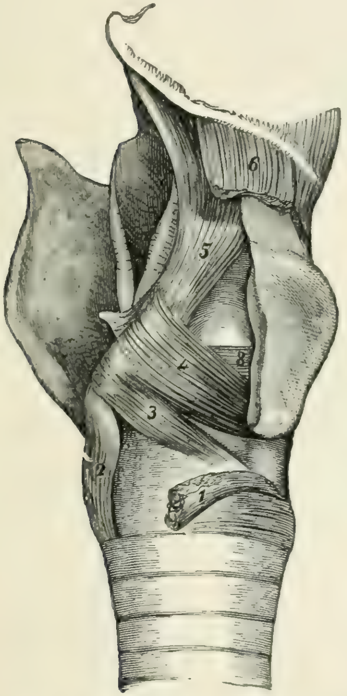


Fig. 135.

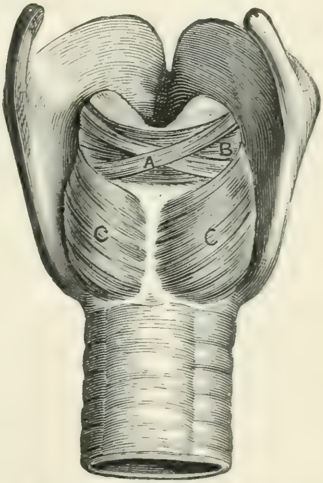


Fig. 134.—View of the internal muscles of the larynx: 1, cricothyroideus detached; 2, crico-arytenoideus posticus; 3, crico-arytenoideus lateralis; 4, thyro-arytenoideus, superficial part; 5, depressor of the epiglottis; 6, thyrohyoideus, cut; 8, deeper part of thyro-arytenoideus. (Ellis.)

Fig. 135.—Posterior view of the larynx: A, superficial part of the arytenoideus muscle; B, deep part of the arytenoideus; C, crico-arytenoideus posticus. (Ellis.)

To the outer side of either cord may be seen a band or fold of mucous membrane known as the **ventricular band,** pocket ligament, or false cord.

The ventricular bands form the superior boundaries of the **ventricular pouches** or pockets.

Both the bands and pouches may be the seat of catarrhal inflammation, œdema, and glandular enlargement.

MALFORMATIONS OF THE LARYNX.

They may be **congenital** or **acquired.**

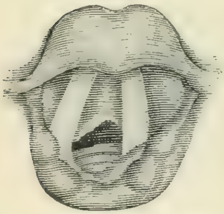
Congenital Malformations.

The **etiology** is but little understood, although it is probable that the condition of the father and mother have more or less to do with it.

Varieties: They may be grouped under three headings, namely:

(*a*) **Congenital stenosis**, which is usually due to a *web* (Fig. 136) stretched across the anterior commissure from cord to cord, or from the ventricular bands. This condition may be present for years without attracting notice, when some intercurrent disease of the larynx brings it under the observation of the physician. The web is usually of the same color as the cords.

FIG. 136.



Laryngeal stenosis due to web between the vocal cords. (Cohen.)

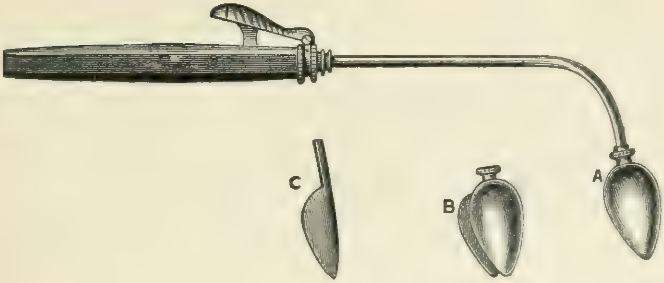
(*b*) Another form of congenital malformation is **laryngeal dilatation,** or **laryngocele.** This is rare in man, but is quite common in some of the lower animals.

(*c*) **True hypertrophy,** or enlargement of the normal tissue, is occasionally found as a congenital condition, although it is usually acquired.

Congenital malformations of the larynx—treatment: If the web causes stenosis of the larynx it may be treated by the introduction of an O'Dwyer tube, leaving it in place for a few hours daily. It may be necessary first to cut the web with Whistler's cutting dilator, shown in Figure 137.

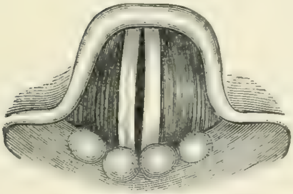
If there is stenosis from *true hypertrophy* (Fig. 138) the enlarged mass should be removed with instruments or caustic applications, as fused crystals of chromic acid, or with the actual cautery. Chemical caustics are to be cautiously applied,

FIG. 137.



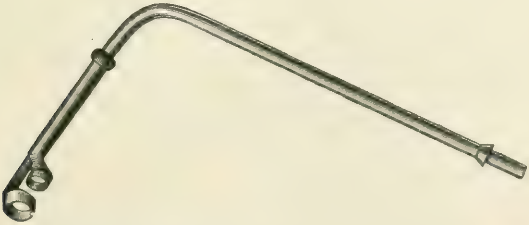
Whistler's cutting dilator.

FIG. 138.



Faulty approximation of the vocal cords as often observed in chronic hypertrophic laryngitis. (Coakley.)

FIG. 139.



Punch forceps for removing laryngeal growths.

as they are apt to excite the formation of cicatricial bands. If the hypertrophy is large enough it may be removed with the biting laryngeal forceps (Fig. 139).

Acquired Malformations.

Etiology: They are usually due to some injury, as: attempted suicide by cutting the throat; the inhalation of a corrosive vapor; or the deglutition of scalding or caustic fluids. Syphilis, lupus, tuberculosis, cancer, and benign neoplasms also give rise to malformations.

Syphilitic Stenosis of the Larynx.

Etiology: The stenosis may be due to œdema; or to cicatricial bands and webs stretching from cord to cord, or between the ventricular bands. Œdema may occur at any period in the course of the disease.

Syphilitic stenosis of the larynx—symptoms: (*a*) Those due to interference with voice-production, as permanent hoarseness and restricted range of register; (*b*) those due to interference with respiration, as repeated attacks of dyspnœa. Such attacks recurring at frequent intervals for a number of years are almost pathognomonic of syphilitic disease of the larynx; (*c*) cough, (*d*) pain, (*e*) dysphagia, and (*f*) expectoration are often present. The expectoration is, however, usually scanty.

Treatment: The usual constitutional remedies (potassium iodide and mercury) should be administered. The iodide should be pushed to the point of toleration. Should alarming symptoms arise from œdema, tracheotomy may become a necessity. It should be done low down so as to avoid the œdematous tissues of the larynx. The tube should be left in place for some days, as its removal might be followed by an extension of the œdema downward, thereby preventing its reinsertion. In the cicatricial type of stenosis the Whistler cutting dilator should be used and the O'Dwyer tube inserted. Daily dilatation with bougies may be practised instead of introducing the O'Dwyer tubes.

Tubercular Laryngeal Stenosis.

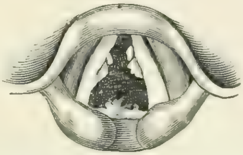
It is usually due to œdema (Fig. 140). Cicatricial stenosis does not occur, as there is little tendency toward a reparative process. The treatment should be as for œdema from other causes.

Lupous Laryngeal Stenosis.

Pathology: The reparative process is prominent, hence *cicatricial bands* are apt to form. They sometimes almost completely close the glottis until the patient has barely enough space left through which to breathe or vocalize. Ordinarily respiration or vocalization are but little interfered with, as the bands are located above the cords.

Prognosis: There is a tendency to spontaneous atrophy of the new-formed tissue, hence the prognosis is more favorable than in other types of stenosis. It should be remembered that there is some tendency toward the true tubercular type, which, if it occurs, renders the prognosis quite grave.

FIG. 140.



Tubercular infiltration of the interarytenoid space with tubercular papillomata of both vocal cords. Characteristic œdematous infiltration of the aryepiglottic folds. (Cohen.)

Lupous laryngeal stenosis—treatment: Surgical measures should not be undertaken unless there is marked embarrassment to respiration. Dilatation is of little value unless preceded by cutting the web. So long as there is active inflammation, radical measures should not be instituted unless there is imminent danger of suffocation.

Traumatic Laryngeal Stenosis.

Etiology: This condition may arise from intentional or accidental wounds to the throat; from foreign bodies; or caustic fluids within the larynx.

The **symptoms** are those peculiar to interference with respiration and vocalization.

The **prognosis** should always be guarded, as there is a tendency for the cicatricial bands to increase and suffocation thereby be produced. Œdema may also develop and cause sudden alarming symptoms. The prognosis as to the voice should also be guarded, as the latter is apt to be seriously impaired.

Traumatic laryngeal stenosis—treatment: The degree of the stenosis and the nature of its cause will determine the treatment. If due to granulation formations these should be removed with laryngeal curettes. If due to œdema, scarification should be done; and, if it fails to afford relief, tracheotomy must be performed. Cicatricial bands should be cut and O'Dwyer's tubes inserted daily.

INFLAMMATIONS OF THE LARYNX.

General Etiology of Inflammations of the Larynx.

Mouth-breathing: Mouth-breathing may be due to enlargement of the pharyngeal and faucial tonsils; retro-pharyngeal abscess; deformities and neoplasms; also to congenital stenosis of the nasal fossæ; hypertrophy and swelling of the nasal mucous membrane; neoplasms; and foreign bodies.

Undue patency and consequent dryness of the nasal fossæ may be due to congenital arrest of development of the turbinal bodies; atrophic rhinitis; the surgical removal of the inferior turbinals; and collapse of erectile tissue of the nose. The effects upon the larynx is much the same as that produced by mouth-breathing.

When the nasal and buccal secretions are *normal* the pharynx and larynx are protected against disease.

Lymphoid glands: The lymphoid tissue of the pharynx, mouths of the Eustachian tubes, base of the tongue, the fauces, the larynx, the nasopharynx and nose, is now described in literature as discrete (scattered lymphatic glands) pharyngeal, tubal, lingual, palatal, laryngeal, and nasal tonsils respectively.

The *function* of the lymphoid glands is to throw out leucocytes on the surface epithelium, where they destroy micro-organisms and other deleterious substances. Inflammations of these structures are usually due to one of two causes: viz., (*a*) microbes; (*b*) diathetic states.

Nasal disease and malformations: Catarrhal inflammations of the throat are usually secondary to some nasal disease or malformation. When there is a fibrinous exudate it is probably of microbic origin and the prognosis should be guarded. Laryngeal inflammations are usually subacute extensions from the nares, and are associated with nasal stenosis. While inflammations of the throat are usually associated with disturbances of the vaso-motor nervous system, microörganisms also act as important causes.

Constitutional conditions: The rheumatic or gouty diathesis plays an important *rôle* in the throat and nose, but more especially in pharyngeal diseases. It has been observed that when certain tonsillar diseases are remedied by local measures, the rheumatic attack sometimes shifts to another part of the body; this should be explained by the migratory nature of rheumatic affections and not be attributed to the local treatment of the tonsils.

Anatomical considerations: *The soft palate and uvula* on account of their intimate relation to the nasopharynx exert a marked influence in the morbid processes of that region.

The *faucial tonsils*, being located between the pillars of the fauces, when adherent or enlarged, interfere with the action of the palatine muscles, and thereby render the voice thick and offer interference to the patency of the Eustachian tubes.

The *laxity of the pharyngeal mucosa* accounts for the frequent occurrence of abscesses in this region.

New formations or *foreign bodies* in the pharynx give rise to pain and interfere with deglutition and vocalization.

Nasal stenosis results in the loss of the respiratory functions of the nose, thereby allowing the inspired air to pass into the lower respiratory tract insufficiently moistened, tempered, and filtered. This results in a train of irritative phenomena already more fully described under *postnasal adenoids*.

In regard to disturbance of the general circulation, it may be said that *plethora* favors local congestion, while *anæmia* predisposes to local muscular relaxation.

Improper use of the voice: Various writers have recently called attention to the fact that the improper use of the voice among singers, public speakers, and criers accounts for most of the morbid conditions peculiar to their professions. Formerly it was held that the local morbid condition was the cause of the vocal disturbance, and that in order to affect a cure, rest of the voice and local, surgical, and medical interference were necessary. In accordance with the present view the rational treatment consists of the proper training of the vocal apparatus, thereby rendering it capable of performing almost any amount of work put upon it.

The influence of improper clothing: The mode of life, habitation, and occupation should be taken into consideration in advising the patient as to the *kind* and *amount of clothing* he should wear. It is common to observe men in rural districts who wear but scant clothing, and who suffer but slightly from catarrhal affections. On the other hand, when the same individuals remove to cities, live in warmer houses, wear heavier clothing, and are less exposed to the weather, it is no uncommon thing for them to develop marked catarrhal diseases of the nose and throat. The amount of clothing should be modified to suit the immediate environment of the individual—*i. e.*, it should be so arranged that when in the house the clothing may be light in weight, conforming to the temperature of the room. While out of doors in severe weather outer coats should be used so as to protect the body in proportion to the change in environment. Especial attention should be given to the feet, as body-temperature may be rapidly lowered by abstracting heat through the soles. In cold weather heavy shoes should be worn, thereby overcoming one of the most fruitful sources of colds and other catarrhal diseases of the nose and throat.

Anæmia of the Larynx.

General anæmia is attended by anæmia of the larynx as a matter of course.

When, however, there is (*a*) *functional aphonia* associated with the laryngeal anæmia; or (*b*) anæmia of the larynx, lower pharyngeal wall, and vocal cords, and no general anæmia, it is pathognomonic of tuberculosis.

The *importance* of this symptom justifies the prominence given it by placing it at the opening of the section on Inflammations of the Larynx.

Acute Catarrhal Laryngitis.

Etiology: *Age*: The larynx is smaller in children, hence is more easily stenosed than it is in adults. While acute laryngitis is more frequent in adults than in children, it is not so dangerous. Enlargement of the postnasal lymphoid tissue before puberty predisposes to laryngeal inflammation, while in later life postnasal catarrh acts as a cause. Plastic exudates are more common in children.

Climatic conditions, as cold, moist air, are exciting causes of catarrhal inflammations here as elsewhere in the respiratory tract. The clothing should be so regulated as to protect without "coddling" the individual.

Improper use of the voice, especially if there are lowered vitality from overwork, anxiety, grief, or sickness, may cause laryngitis.

Poor ventilation in shops, school-rooms, stores, and bedchambers predisposes to attacks of acute laryngitis. Fumes, gases, and dust which irritate the larynx should be carried off with ventilating currents of air.

Disturbances of digestion and of the portal circulation which arise from the abuse of alcohol, tobacco, and food predispose to hyperæmia and acute inflammation of the larynx.

Acute catarrhal laryngitis—symptoms: These will vary according to the location and amount of activity of the inflammatory process. If the aryteno-epiglottic folds and epiglottis are involved, as in laryngitis due to scalding drinks or liquors, *dysphagia* will be a prominent symptom. If the true cords and ventricular bands are prominently involved, there will be *dysphonia*. Should the interarytenoid space be the seat of inflammation, *cough* will be the prominent symptom. The cough may have a clear tone and the voice be almost free from hoarseness. In many of the so-called dry coughs attributed to chronic bronchitis, if the interarytenoid space (posterior commissure) were examined laryngoscopically it would be found to be the seat of inflammatory thickening. *Hoarseness* is more or less prominent according to the amount of thickening of the vocal cords and impaired action of the laryngeal muscles. *Œdema* and *hemorrhage* are more apt to occur in laryngitis of singers and public speakers than in persons of other callings. This is perhaps due in part to excessive use of the voice after the inflammatory process is established.

Age also modifies the symptoms, as in children there is a greater tendency to *spasmodic closure* of the glottis than in adults. This is especially true between the ages of two and five years. This may be accounted for in several ways, viz.: at that age there is a predisposition to the spasmodic action of the muscles from reflex irritations. The larynx is much smaller in the child than in the adult, hence the dried secretions which accumulate during sleep, especially if the child is a mouth-breather, may be sufficient to obstruct the glottic opening. I have observed in children affected by lymphoid enlargements, as postnasal adenoids and hypertrophied faucial and lingual tonsils, that *pseudocroup* commonly forms part of the clinical history of the early part of their lives. This may be accounted for by the obstruction to the circulation afforded by the lymphoid masses, and by the fact that nasal respiration is interfered with. The child's larynx is thus subjected to dry and irritating air which lowers the vital resistance of the laryngeal tissues and renders the secretions abnormally heavy and tenacious.

The *constitutional symptoms* are about the same as those in acute rhinitis and need not be detailed here.

Acute catarrhal laryngitis—laryngeal image: Great variation will occur in the laryngeal image according to the particular location of the inflammation, the age of the patient, his occupation, and the stage of the disease.

Small areas or the whole membrane may be slightly thickened or red, the cords may remain a whitish-yellow, being little or not at all thickened; or they may be quite red and swollen. At the onset the membrane is dry, but at the end of twenty-four hours a glairy mucous secretion is present. In some cases after a few days the secretion may assume a mucopurulent character. The mucous membrane, which at first presented an evenly red and swollen appearance, may in the later stages present patches of softened and desquamated epithelium. The submucous tissues may also be broken down beneath these spots. The ventricular bands may be so swollen that they overlap the true cords and interfere with phonation.

Prognosis: Rest and the proper hygienic surroundings, if complied with at an early stage, will in most cases effect a spontaneous cure.

Acute catarrhal laryngitis—treatment: The patient should be positively instructed not to use his voice even in a whisper for a period of forty-eight hours. All communications should be made by signs or in writing, and he should be confined within the house where the temperature is maintained at about 65° to 70° F. He should be not only confined to the house, but within a certain room of the house, in which the atmosphere is kept heavily laden with moisture from boiling water. He should be taught that absolute relaxation of the vocal apparatus, and, indeed, the whole body, will aid materially in restoring his voice to perfect condition within a very short time.

In addition to the foregoing requirements, he should wear a wet compress, made of a small piece of folded linen, on his neck over the larynx. The compress should be held in place by a band of flannel a little wider than the compress and long enough to go twice about the neck. The compresses should be wet in cold water every four or five hours, squeezed slightly to avoid dripping, and applied as above described. Twice a day they should be moistened in the following solution:

℞. Tr. iodin., ʒss;
Glycerin., ʒiv;
Aquæ, q. s. ad ℥iv.—M.
Sig. Use twice daily to wet compress.

Four or five ¼-grain doses of calomel should be given at intervals of two hours, after which free purgation should be produced by the administration of a full dose of Glauber or Epsom salts.

If the above conditions are not complied with, the disease may run a course covering from one to three weeks and render the patient very liable to subsequent attacks, if not to permanent chronic laryngitis.

As violent coughing aggravates the inflammation, it should be allayed by the administration of small doses of morphine or codeine. Should the secretions be very heavy and tenacious, the administration of $\frac{1}{100}$ grain of pilocarpine with very minute doses of antimony and ipecac is advisable.

Should it be thought necessary to produce emesis, one drachm of sodium chloride in a half tumbler of water, followed by a copious drink of hot water, will produce the desired effect with the least possible harm.

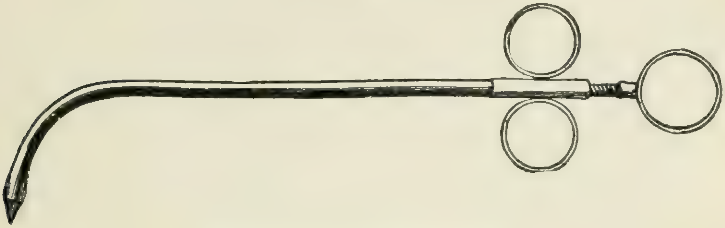
If there is considerable tenderness over the larynx, the parts should be massaged freely with camphorated oil.

Singers and public speakers, and others who specially prize their voice, should make an annual visit to the sea or mountains for the benefit to be derived from the pure air, rest, and change of environment. All patients should be instructed as to the necessity of protecting the feet from wet and cold by wearing heavy boots. A cold sponge-bath, followed by vigorous friction, should be indulged in every morning. In this way the activity of the skin will be maintained and the probability of future attacks be very much lessened.

Other remedies of more or less value may be used according to the indications in the individual case. In the pseudo-croup type the patient should be put into a warm bath and cold douches applied to the spinal region. The baths should be continued for about ten minutes. One teaspoonful of the compound tincture of benzoin in one-half pint of boiling water may be used at intervals of from two to three hours as a steam inhalent to relieve cough and irritability. Bland sprays of oil containing small quantities of the oil of pine-needles and the oil of tar are of some value. Kyle reports favorably on the use of dilute nitric acid in five- to ten-drop doses in water every half-hour for three doses, as a means of relief for those who are compelled to use their voices. The cold water or ice-bag applied over the neck in the early stages often relieves the distressing symptoms.

The surgical treatment: Should there be moderate œdema of the epiglottis or mucosa of the larynx, they should be freely scarified with a Tobold concealed laryngeal lancet (Fig. 141). There is little danger of serious hemorrhage from this procedure, as the parts are filled with serous fluid rather than blood. Scarification should be followed by an application of mild astringents, as the glycerite of tannin. This form of astringent is especially valuable, as the glycerine promotes osmosis of serum from the œdematous tissues, while it carries the tannin into the tissues. When there is imminent danger of suffocation, intubation or tracheotomy may be indicated to prevent a fatal issue.

Fig. 141.



Tobold concealed laryngeal lancet.

Laryngitis Symptomatic of Constitutional Diseases.

Erysipelas: In the early stages it resembles simple acute laryngitis. Later it extends to adjacent parts, sometimes even involving the face. Other exposed persons may become similarly affected, and finally a bacteriologic examination will reveal the characteristic germs of erysipelas. In the severer types the larynx may be affected by a phlyctenular or even a herpetic eruption. Rarely gangrenous areas develop.

Measles: Usually there is a mild profuse redness with hoarseness and aphonia. A careful laryngoscopic examination will show the characteristic rash and swollen follicles. Should the voice be used in crying or talking there may be frictional erosions. There is usually a barking cough, which in a few days is followed by dysphonia.

The *treatment* should consist of a massage of mercurial ointment over the larynx, wet compresses, and cathartics. In rare cases it may be necessary to resort to tracheotomy.

Scarlet fever: Laryngitis is less frequent in this disease, and is attended by no symptoms of especial importance.

Smallpox: Small whitish prominences composed of necrotic areas of epithelium, under which there is a small amount of pus, form the chief objective symptom.

Typhoid fever: There is a deposit of specific bacilli, typhoid infiltration, and ulceration of the mucosa. The edge of the epiglottis is subject to erosions which may leave scars. They may in time produce laryngeal stenosis.

Influenza: The whole respiratory tract is involved. The interarytenoid mucosa is especially prone to involvement, which accounts for the dry barking cough so common in this disease. It so nearly resembles whooping-cough that a mistaken diagnosis is often made. Laryngeal hemorrhage may occur. The true vocal cords may become eroded. Although the disease is quite active, abscess-formation is rare.

Whooping-cough: The anterior surface of the posterior wall of the pharynx and larynx are red and slightly swollen, hence the constant source of irritation which expresses itself in the form of a series of spasmodic coughs. The secretion is scanty and difficult to remove. Ecchymosis and hæmatoma are sometimes found in the submucous tissue. *Local treatment* is of little value.

Rheumatism: There is violent pain in the arytenoid and other cartilages. The arytenoid cartilages are restricted in their movements, hence vocalization is more or less interfered with. There is swelling of the mucous membrane covering the cartilages. The presence of pain, the restricted use of the muscles of adduction and abduction, and pain and swelling over the arytenoids form a group of symptoms which should not be mistaken for true paralysis.

Typhus fever: Laryngeal inflammation sometimes occurs, and may be quite dangerous. The membrane is bright red or dark and velvety, and covered with a sticky mucopus. Deep ulceration may expose the cartilages.

Miasmatic epiglottitis: Marked œdema, dysphagia, odontophagia, and dyspnœa are the chief symptoms. The disease is usually found among people living in salt marshes.

Malarial poisoning, characterized by intermittent fever, hoarseness, redness, œdema, and dyspnœa, has been observed and reported. Should the glottis be markedly œdematous it should be scarified, and cold compresses applied to the neck. Iced astringent sprays should be thrown into the larynx. The usual antimalarial treatment should be faithfully carried out.

Laryngismus Stridulus.

Etiology: This is a disease characterized by spasm of the glottis, accompanied by stridor. Disease of the larynx and trachea, whether it be inflammatory or non-inflammatory in character, predisposes to this condition. Many cases have been noted in which intestinal disorder seems to be the only assignable cause. Likewise the phenomena occurring during dentition in early childhood holds a close relation to the disease. Rickets, neuroses, faucial disease or tumor, nasopharyngeal adenoids, uterine disease, the uric-acid diathesis, elongated uvula, and necrosis of the vertebræ, may, through circulatory changes and reflex irritations, cause laryngismus stridulus. Pressure upon the pneumogastric or spinal accessory nerves may bring about very serious consequences.

The **prognosis** may be quite grave, while in other cases the danger is remote. The treatment, however, should always be promptly and vigorously prosecuted, and in the interval between attacks the special causes be sought for and removed if possible. One attack is quite apt to be followed by others.

Laryngismus stridulus—treatment: The first consideration should be the prompt relief from the spasmodic seizure. The child should have cold water splashed on his spine and hot water on the nape of the neck. The tongue should be seized with a napkin and pulled upon rhythmically one-fourth as many times as there are pulse-beats, in order to excite the respiratory centres. If the spasm is not speedily relieved, the larynx should be examined for foreign bodies or other sources of irritation. The administration of from five to ten drops of ether will sometimes afford prompt relief. The mucous secretions should be removed with the finger or probang. The administration of oxygen under pressure is attended with very favorable results, as asphyxia is imminent. After a warm bath of ten minutes' duration, the child should be wrapped in a warm blanket and wiped dry with a towel introduced within the blanket. After the acute attack has subsided the patient should be carefully examined for postnasal adenoids or enlarged faucial and lingual tonsils. Indeed, the entire system should be subjected to a most careful examination by the best practitioners and specialists available. This is justified on account of the probable return of the disease and the serious nature of its causes.

Traumatic Laryngitis.

Etiology: There is no difference between this form of laryngitis and the usual acute type, except its etiology. As its name implies, it is due to some form of external injury, as attempted suicide by cutting the throat; violent contusion from some external source; accidental or intentional swallowing of caustic fluids; the inhalation of irritating gases and vapors; or to meddlesome or awkward topical treatment. If due to scalds or caustic fluids the adjacent tissues will be involved, and dysphagia be a prominent symptom.

Treatment: If there is sufficient œdema to cause stenosis of the larynx, scarification, intubation, or tracheotomy may become necessary.

Acute Epiglottitis.

Occurrence: This condition is usually associated with inflammation of the adjacent tissues. The lingual tonsils are most often simultaneously inflamed.

The **treatment** is as given for acute laryngitis. Here, as in laryngitis, it is quite important that the nasal and postnasal spaces should be freed from obstruction or other diseased condition that interferes with their respiratory functions.

Membranous Laryngitis (Membranous Croup and Exudative Laryngitis).

Definition: This is a form of membranous laryngitis not caused by the Klebs-Löffler bacillus. It is not infectious, and therefore is never an epidemic disease.

Etiology: It is usually preceded by an exudate upon the fauces. If the exudate is upon the surface of the tonsils it is more apt to cause membranous laryngitis than if it is within the crypts. The disease may be sporadic or endemic, but never epidemic. Previous inflammations of the larynx, and especially pre-existing nasal catarrh, favor the development of the process. Sanitary and hygienic conditions have more or less to do with the causation. Improper clothing and living in damp, cold rooms are especially fruitful of this type of inflammation.

There has been considerable controversy as to whether membranous laryngitis is not always a true diphtheria. The Klebs-Löffler bacillus is sometimes found in the false membrane, while the clinical history of the case presents none of the characteristic signs of diphtheria. On the other hand, the membrane is sometimes found to be entirely free from diphtheritic bacilli, while profound toxæmia is present. We are hardly prepared to make a positive differentiation between the two diseases, and yet, for clinical purposes, it seems well to describe them separately.

Membranous laryngitis—pathology: At the onset the membrane is hyperæmic and slightly swollen, as in an acute catarrhal process. At the end of eighteen to twenty-four hours the exudate begins to coagulate upon the surface, the rapidly proliferating epithelium and leucocytes being entangled within its meshes. The psuedomembrane thus formed usually involves only the epithelial layer, the submucous tissue being unaffected. Occasionally, however, the submucous tissue breaks down, but this is probably due to interference with the circulation of the parts rather than to an extension of the plastic exudate beneath the epithelium. The exudate is usually above the glottis, and after it is well developed, is situated mostly on the true cords. As it travels downward from the fauces it attacks the epiglottis and ventricular bands before reaching the true cords.

Membranous laryngitis—symptoms: The febrile movement usually begins with a chilly sensation or a distinct chill, the temperature ranging from 100° to 104° F., the pulse being full and bounding. Anorexia and restlessness are early manifestations. It may thus be seen that the febrile movement is quite different from that of simple catarrhal laryngitis, assuming more nearly that of true diphtheria, although the temperature may range even higher. The patient complains of the throat feeling stiff and sore, especially upon deglutition. The pains radiate from the larynx toward the ears. The temperature-index does not change materially during the first twenty-four to forty-eight hours, but as the larynx becomes covered with the membrane, it rises one or two degrees. The voice is quite characteristic, having a metallic ring or bark upon coughing or attempted vocalization, and may become completely aphonic. This symptom will vary with the amount and location of the false membrane and with the degree of muscular impairment. Dyspnœa is present in proportion to the amount of laryngeal stenosis, and is both inspiratory and expiratory. It is attended by the usual signs, as cyanosis, supraclavicular depression, anxious expression of countenance, extreme restlessness, and dilated nostrils. It is progressive until death ensues or the membrane is thrown off. The membrane may re-form, hence extreme care should be observed until convalescence is well established. The natural course of the disease is from three to six days, the larynx being invaded about the second or fourth day, when the suffocative symptoms appear. The presence of albumin in the urine is not significant of true diphtheria, as it may be found in simple follicular inflammation of the tonsils. The same may be said of paralysis of the palatine muscles.

Membranous laryngitis—diagnosis: The only disease with which it may well be confounded is diphtheria; and as the presence or absence of the Klebs-Löffler bacillus does not afford a positive means of diagnosis, it is well to give the patient the benefit of the doubt and treat him as for diphtheria. Bacteriologic examination of the secretion and fragments of the membrane should be made early in the disease, but failure to find them should not be taken as sufficient data upon which to discard diphtheritic treatment.

Membranous laryngitis—treatment: In the early stages of the disease, before the formation of croupous membrane, *cold* applied to the outside of the neck will sometimes abort the disease. It may be applied by means of a long, narrow ice-bag partially filled with cracked ice, or by means of a Leiter coil made of lead tubing so bent as to conform to the shape of the neck. If the ice-bag is used, it should be wrapped with a piece of thin muslin, and should be refilled every hour. If the Leiter coil is used, it should be connected by means of a siphon tube with a basin of ice water placed on a higher level than that of the patient. In this way the neck is subjected to the action of the continuous current of cold water. Heat may be used by either of the above methods, but is ordinarily less efficacious than cold.

The *atmosphere of the room* should be kept at a temperature of about 75° F. and surcharged with lime vapor. A good method of administering lime vapor is to slack a pound or two of lime in a vessel of water by the side of the bed, a temporary tent, made of a sheet, being so held as to cover the child and lime-vessel. The child should be thus compelled to inhale the vapor of the lime for ten minutes, at intervals of one to three hours. The particles of lime loosen the pseudomembrane and bring the disease to a speedy and favorable termination. If lime is not available, the air of the room should be surcharged with steam from boiling water to which have been added carbonate of sodium and spirit of turpentine. Steam may also be administered directly to the child by means of an atomizer placed on a stand by the side of the bed. These and other remedies, as the compound tincture of benzoin, creosote, oil of pine-needles, oil of eucalyptus, and the benzoate of sodium, may be thus administered for the relief of cough and the thick, tenacious secretion.

Internal medication: In this, as in exudative inflammation of the tonsils, the mixture containing two parts of the tincture of chloride of iron and eight parts of glycerin should be given in half-drachm doses every two hours to a child five years old. The iron thus administered will not disorder the stomach, but will act as a local astringent, and in some way so modify the blood as to restrict its tendency to form the plastic exudate. Calomel should be administered every two hours in one- or two-grain doses until the stools are marked by a greenish color, after which it should be given three or four times daily. In this way from twenty to forty grains of calomel may be administered without producing any deleterious effects, but, on the contrary, producing very favorable ones. After the stools show the greenish color a mild saline purgative should be administered once daily until convalescence is established.

Should laryngeal stenosis become so marked as to produce profound dyspnœa and cyanosis, either *intubation* or *tracheotomy* should be immediately performed.

Perichondritis.

Definition: This is an inflammation of the laryngeal cartilages which may be characterized by ossification, hypertrophy, fibrous thickening, ulceration, or abscess.

Etiology: It is usually due to syphilis, tuberculosis, specific fevers, rheumatism, and traumatism. Children and young adults are rarely affected, it being most common after middle life. Men are more often affected than women.

Perichondritis—pathology: In the early stages there are the usual inflammatory changes, which finally terminate in ossification, hypertrophy, hyperplasia, ulceration, or abscess. Sometimes the opening of the ulcer into the cavity of the larynx is quite small, while the tissues beneath are undermined in all directions for quite a distance. The amount of damage done cannot always be estimated by the apparent size of the ulcerous opening. In all cases the muscular movements of the larynx are interfered with both on account of the articular changes, and the changes within the muscles themselves.

Perichondritis—symptoms: The acute stage is ushered in with a febrile movement as in acute simple catarrhal laryngitis. There are chilly sensations, malaise, headache, loss of appetite, pains in the bones, and a temperature of 100° to 101° F. The vocal functions are impaired according to the degree and location of the inflammation.

In the syphilitic type there are marked tenderness and pain upon pressure. If the cricoid cartilage is involved, the perichondrium may be distended inward, closing the lumen of the larynx, thereby producing dyspnœa. When the posterior part of the cartilaginous box is inflamed, dysphagia becomes a prominent symptom on account of the close proximity to the œsophagus. Important laryngeal muscles may become infiltrated, and the voice impaired or lost. Should one of the arytenoid cartilages be the seat of inflammation, laryngoscopic examination would show a one-sided tumefaction. This is a common location in tubercular perichondritis, the voice becoming hoarse and low in pitch. When the thyroid is involved the voice becomes hoarse or aphonic, and the respiration dyspnœic. As the epiglottis is a fibrocartilage, it is rarely the seat of perichondritis. When the perichondritis assumes the ulcerative form there is a tendency to the formation of a fistulous opening outward through the tissues of the neck, or internally into the larynx or adjacent tissues. The necrotic cartilage is exfoliated through the fistula thus formed.

Laryngeal perichondritis—prognosis: The course is usually a chronic one, and may lead to death by suffocation. In the acute type the attack is sudden and life may be threatened within a few hours. The prognosis is more or less affected by the constitutional dyscrasia causing the disease.

Perichondritis—treatment: The treatment should be varied according to the etiology of the particular case. In a general way it may be said that antiphlogistic measures should be instituted, as cold applications, leeching, wet cupping, etc. If there is a sluggish portal circulation, calomel should be administered until catharsis is produced, when a saline draught should be given. In the syphilitic type the iodide of potassium and other suitable remedies should be given. Tubercular inflammation should be treated by the internal administration of three-grain doses of carbonate of guaiacol every three hours. If there are marked signs of general constitutional disturbance, codliver-oil should be administered in conjunction with a nutritious diet. In the ulcerative type there should be an attempt to stimulate nutrition and assimilation, hoping thereby to arouse the reparative energy of the blood and cellular tissues. Rheumatic inflammations should be treated by the administration of carbonate of guaiacol and antilithæmic remedies.

Where there are ulceration and exfoliation of the cartilage surgical interference may be demanded. The exfoliated cartilage should be removed at once, either through the tissues of the neck or through the mouth. Tubercular ulcers should be curetted and rubbed with lactic acid. If marked dyspnœa arise, intubation or tracheotomy should be performed.

Chronic Catarrhal Laryngitis.

Definition: This is superficial inflammation of the mucosa, characterized by hoarseness or aphonia.

Etiology: This disease is rarely primary, but is nearly always secondary to some form of nasal disease or obstruction, or to disease of the nasopharynx. It is very important that this part of the etiology be well understood, as the principal part of the treatment is based upon it. Any condition of the nose which interferes with the respiratory functions will bring about a lowered power of resistance of the laryngeal mucous membrane, thereby making it a favorable location for catarrhal inflammation. The respiratory functions of the nose being lost or impaired, the inspired air passes to the larynx insufficiently warmed, humidified, and filtered, and irritates the larynx and lower air-tract by overtaxing its glandular and circulatory apparatus. The conditions of the nose which impair its functions may be obstructive, as hypertrophic rhinitis, cystic or polypoid degeneration of the middle turbinal, neoplasms, rhinoliths, foreign bodies, and deflection of the septum. Diseased conditions which are not obstructive, as simple dry catarrhal rhinitis, atrophic rhinitis, and ozæna, may also impair or destroy the respiratory functions of the nose. Hence it is important that intranasal conditions should be carefully examined for when trying to determine the causes of chronic catarrhal laryngitis. In suppuration of the posterior ethmoidal cells and sphenoidal sinuses the pus discharges over the middle turbinal into the nasopharynx, and drops down upon the laryngeal mucosa, where it acts as an irritant. It may thus be the chief cause of laryngitis. Postnasal catarrh also causes laryngitis in the same way. Other causes which are more or less responsible for chronic laryngitis are improper clothing, unhygienic environment, unfavorable climatic conditions, the rheumatic diathesis, the excessive use of alcohol and tobacco, and repeated attacks of acute laryngitis.

Œsophagitis and gastritis have been known to cause chronic laryngitis. Sluggish circulation in the portal system causes a hyperæmia of the upper respiratory tract, thereby favoring the inflammatory process.

Chronic catarrhal laryngitis—symptoms: The most characteristic symptom is the appearance of hoarseness in the morning after very moderate use of the voice. During the day the voice may regain its clearness, but soon relapses into the hoarse condition. Toward evening, when the vital energies are at a low ebb, the hoarseness is especially marked and annoying. Public speaking is attended or followed by hoarseness or complete loss of voice. The amount of hoarseness is somewhat dependent upon changes in the weather. The patient complains of a slight sore feeling in the upper part of the laryngeal box, and will sometimes suggest that it is rheumatism. There is a sensation of "a web-like formation over the vocal cords, which, if removed, would render the voice quite clear." The patient unconsciously attempts to remove the imaginary web by clearing the throat. It should be explained to him that by so doing he gets no relief, but, on the contrary, increases the irritation and perpetuates the laryngitis. Those who have impairment of the nasal respiratory functions may be markedly neurotic; hence in chronic laryngitis the nervous element may be of considerable importance, and should receive appropriate treatment. After slight use of the voice the patient has a feeling of muscle-tiredness. Upon laryngoscopic examination the mucosa appears thickened, sometimes in its whole surface, and at other times in limited areas only.

The foregoing symptoms vary somewhat with the location and extent of thickening. If the interarytenoid space is thickened, a dry cough accompanies the hoarseness.

Chronic catarrhal laryngitis—diagnosis: Some of the symptoms of this disease are common to tubercular, œdematous, paralytic, syphilitic, and malignant disease of the larynx.

Œdema of the larynx presents a membrane of much the same color, but more swollen, with a history of sudden onset and dyspnœa.

Paralysis of the vocal cords is attended by inability to move the arytenoid cartilages and a foul-smelling secretion.

Tubercular laryngitis presents an ashen-colored membrane, with the characteristic history and temperature-index of tuberculosis. There is constant pain in the throat, which is much increased upon deglutition.

In *syphilitic laryngitis* the history and presence of the diffused œdema of the larynx, and a favorable response to the iodide of potassium, render the differential diagnosis comparatively easy.

In *malignant disease* of the larynx the diagnosis in the early stage is rendered somewhat difficult on account of the late appearance of glandular enlargement, œdema, and ulceration. A careful examination of the exterior of the larynx will, however, reveal an enlargement upon one or both sides, while the breath gives a characteristic offensive odor, which, with a history of advanced age, sharp, lancinating pains, laryngeal hemorrhage, and paralysis of the laryngeal muscles upon one or both sides, affords sufficient data upon which to make a positive diagnosis.

The **prognosis** of chronic laryngitis, so far as the recovery of the voice is concerned, should be guarded, as a favorable termination depends very much upon conditions over which the physician has no control. If the cords have not become markedly thickened, and the patient will submit to the surgical treatment needed to restore nasal respiration, and will give his vocal apparatus the needed rest, there is a very good chance for the restoration of his voice. Although the hoarseness seems to be due to a simple catarrhal laryngitis, the prognosis should be guarded if there is a family history of tuberculosis. In many cases the voice remains permanently hoarse in spite of all forms of treatment.

Chronic catarrhal laryngitis—treatment: After giving attention to the clothing, diet, bowels, vocation, and special dyscrasiæ, the intranasal disease should receive appropriate treatment. The nervous phenomena should also be modified by the administration of the bromide of potassium, or five-grain doses of chloral hydrate three times a day. *Local palliative treatment* in the form of astringent inhalants or atomized fluids, containing benzoin, oil of pine-needles, eucalyptus, and camphor, should be used to allay cough, sense of irritation, and cobweb sensation in the larynx. *Local painting*, spraying, or nebulæ, with ten- to twenty-grain solutions of the mineral astringents, as the nitrate of silver, sulphate of copper, and chloride of zinc, exert a favorable influence upon the progress of the disease. The tenacious secretions may be somewhat thinned by the administration of five-grain doses of the benzoate of sodium three times a day. Should the larynx be dry, the administration of the compound wine of iodine, which is composed of iodine, grain $\frac{1}{8}$, phosphorus, grain $\frac{1}{100}$, bromine, grain $\frac{1}{8}$, in sherry wine, will stimulate the glandular activity, thereby affording much comfort to the patient. The application of a compress moistened with a solution composed of iodine, 1 drachm, glycerin, 4 drachms, and water sufficient to make 3 ounces, exerts a very favorable influence. The use, or at least the abuse, of alcohol and tobacco should be interdicted.

To *recapitulate*, the larynx should be given a rest, the nasopharyngeal disease removed, distressing symptoms palliated by the proper use of local remedies, nervous symptoms and constitutional disorders corrected, and, most important of all, obstructive and diseased conditions of the nose should be radically corrected.

Follicular Laryngitis.

Definition: This is a type of chronic catarrhal laryngitis in which there is hypertrophy of the acinotubular glands and the lymphoid tissue of the laryngeal mucosa.

Occurrence: The condition is comparatively rare, and it is quite similar to follicular pharyngitis. It is most common in those who make a professional use of the voice, and it has often been called "preachers' laryngitis."

Treatment: The administration of small doses of the syrup of the iodide of iron or the compound iodine wine stimulates the mucous glands, thus relieving the congestion and thinning the secretions. The patient should be taught the proper use of the vocal apparatus and cautioned against its abuse.

Laryngitis Sicca.

Etiology: This condition is one which is usually secondary to some disease of the nose or pharynx. Atrophic rhinitis is the most common cause. Deflection of the septum, tumors, ridges, and spurs may also cause it. Dry laryngitis is more common in females. A dry pharyngitis may also be associated with laryngitis sicca.

Laryngitis sicca—symptoms: During waking hours the movements of the vocal apparatus keep the crusts from accumulating, hence the symptoms are comparatively slight. On waking of mornings there is more or less loss of voice on account of the presence of the crusts and inspissated mucus.

If the crusts are large, there may be pronounced dyspnœa. Their presence causes a sense of irritation and cough. Upon inspection with the laryngoscopic mirror greenish-yellow crusts may be seen below the cords, although they are occasionally above the cords. The breath is fetid upon *oral* exhalation. It is necessary to make a distinction between oral and nasal exhalation, as the nose imparts its fetid odor in nasal exhalation. Sometimes there are erosions of the mucosa under the crusts; hence when the crusts are expelled by coughing there may be slight hemorrhage.

Prognosis depends largely upon the curability of the nasal and pharyngeal diseases causing it. If the atrophic rhinitis is in the early stages, there is a fair chance of effecting a cure; if, however, it is well advanced the prognosis is bad. If it is due to deflection, spurs, ridges, or intranasal tumors, the prognosis is good after the removal of such, provided that the glandular elements are not too badly diseased. Pharyngitis sicca is rather intractable to treatment; therefore a dry laryngitis secondary to it is also intractable.

Laryngitis sicca—treatment: It is of prime importance that the intranasal disease causing it should be remedied. The atrophic rhinitis, septal abnormalities, tumors, etc., should be corrected if it is possible so to do. Any diseased condition of pharynx and nasopharynx should also receive appropriate treatment.

The *local treatment* of the larynx should consist of detergent sprays (Dobell's or Seiler's solution) at intervals of three or four hours. This may be done by the patient. The physician should make applications of astringent and slightly stimulating lotions to the larynx. For this purpose weak solutions of silver nitrate, sulphate of zinc, compound tincture of benzoin, etc., may be used. The treatment is usually a protracted one.

The internal administration of the iodide of potassium in four-grain doses three times a day, or the compound wine of iodine three times a day in plenty of water, will stimulate the mucous glands and render the disease more tolerable.

A *change of residence* to a climate with a uniformly humid atmosphere affords great comfort.

Hypertrophic Laryngitis.

Etiology: This is a comparatively rare disease, and is the result of prolonged over-nutrition of the parts from persistent hyperæmia.

Pathology: It has been described by some writers as a scleroma, but a true scleroma is characterized by an excess of fibrous tissue, attended by ulceration and contraction; while in true hypertrophy all the elements of the mucous membrane are increased and there is no tendency to contraction.

Symptoms: The movements of the vocal cords are interfered with according to the location and degree of hypertrophy. If it is over the arytenoid cartilages, vocalization is impaired, while respiration may remain perfectly free. Should, however, the hypertrophy involve the ventricular bands to such a degree that they project over the vocal cords, not only will vocalization be impaired, but respiration as well. The hypertrophic process, however, is located most often in the posterior commissure, where it may be seen as slightly nodular or granular eminences, projecting upward and forward into the chink of the glottis (Fig. 138). When in this location the motility of the cords is somewhat impaired and cough is a more or less prominent symptom. Œsophagitis sometimes causes this condition by an extension of the inflammatory process, and should, therefore, receive appropriate treatment.

The **treatment of hypertrophic laryngitis** is of comparatively little value. If the hypertrophy is excessive, it may be removed by means of laryngeal cutting-forceps (Fig. 139), chromic-acid applications, or the actual cautery. Careful examination should be made for the causes of the condition and measures addressed to their relief, hoping thereby to arrest the progress.

Œdematous Laryngitis (Phlegmonous Laryngitis; Submucous Laryngitis.)

Definition: This is a form of laryngitis attended by a serous infiltration of the submucosa of the larynx and epiglottis. Occasionally the mucosa of the trachea immediately beneath the cords is also involved.

The *predisposing causes* are (*a*) a mild acute catarrhal laryngitis; (*b*) male sex; (*c*) early adult life; (*d*) general debility; and (*e*) great fatigue.

The *exciting causes* are "colds"; draughts; exanthematous fevers; irritating vapors; steam inhalations; foreign bodies; syphilitic or tubercular infections; and retropharyngeal abscess.

While it is usually found in young adults, it occasionally occurs in infants and octogenarians.

Pathology: The inflammatory products of acute laryngitis, plus vascular turgescence and serous infiltration of the loose submucous tissue, are present. The parts most affected, therefore, are the aryepiglottic folds, ventricular bands, and the posterior surface of the epiglottis. The cords are usually free from infiltration, but are red from vascular congestion. The swelling is usually symmetrical. During the late stage of the disease there may be pus formation in the submucous tissue.

Œdematous laryngitis—symptoms: It is generally ushered in by chilly sensations or a distinct chill. A febrile movement of 100° to 103° F. is established. Dyspnœa immediately follows the onset of the fever, and it may progress gradually or develop suddenly until extreme distress and danger to life are imminent. The voice is rough and heavy, while respiration is difficult. In the beginning the obstruction to the breathing is inspiratory. Later the expiratory effort is also obstructed. Extreme anxiety then becomes a prominent symptom. The dyspnœic symptoms last for about twelve to seventy-two hours. Cough is incomplete and ineffective. Deglutition may be difficult and distressing. The appearance of the œdematous and inflamed laryngeal mucosa depends somewhat upon the exciting cause of the disease. The peculiar eruption of the exanthem, the character and nature of the traumatism, will modify the appearance of the infiltrated mucosa. In a general way the infiltrated and inflamed mucosa presents a globular semitransparent appearance with a bright-red circumference. The capillaries of the epiglottis are injected and the vocal cords are red and somewhat thickened. When the membrane below the glottis (infraglottic) is œdematous, it is still more red than the cords.

Prognosis and course: The disease usually develops rapidly, and may lead to a fatal issue within a few hours. The dyspnœa generally, however, lasts for three or four days. The danger is almost always from asphyxia. Occasionally an abscess or gangrenous process leads to a fatal termination. Œdema of the infraglottic mucosa renders the prognosis very grave.

Œdematous laryngitis—treatment: The most important indication is to overcome the dyspnœa and thereby avoid suffocation. The most efficient method of accomplishing this is by means of Tobold's concealed laryngeal lancet (Fig. 141). The scarification should be repeated three or four times a day to unload the serum from the distended membrane. Some benefit may be derived from the use of steam inhalations and pellets of ice in the mouth. Leeches applied over the cricothyroid membrane, and the ice-bag or the Leiter coil applied continuously are valuable adjuncts to the treatment. If pus is present, it should be evacuated. If the dyspnœa is progressive and the patient becomes cyanotic, tracheotomy should be done. It should be done low down on account of the possibility of there being infraglottic œdema. After the tracheotomy-tube is introduced it should be allowed to remain as long as there seems to be œdema, as the parts about the wound might become œdematous in its absence and render its reintroduction difficult or impossible.

Œdema of the Larynx.

Definition: Simple œdema of the larynx is unattended by true inflammation as is œdematous laryngitis. It is a serous infiltration of the loose submucous tissue in the same anatomical regions affected in œdematous laryngitis.

Etiology: It is usually due to the same causes that produce œdema in other parts of the body. Some form of kidney disease is the most common cause. Cardiac weakness or insufficiency, vasomotor disturbance of the capillary circulation, tuberculosis, syphilis, traumatisms, irritating air, and gases may also cause it.

Œdema of the larynx—symptoms: The onset is sudden, and the signs are those of inspiratory obstruction. This gradually increases with intervals of comparative comfort for two to four days, when a fatal issue may occur if the remedies prove ineffectual. The dyspnœa is accompanied by stridulous inspiration. After the first six to twelve hours the dyspnœa becomes quite marked, hoarseness and aphonia develop, the patient becomes restless and assumes an anxious expression of countenance. There is a sense of fulness and tension in the throat, with slight pain upon swallowing. The *laryngoscopic appearance* is quite similar to that of œdematous laryngitis except the membrane is more translucent and less red.

Œdema of the larynx—prognosis: The prognosis depends somewhat upon the cause of the local disease. If it is due to simple catarrhal conditions of the pharnyx and upper air tract the prognosis under treatment is very good. If it is due to syphilis or tuberculosis the prognosis as regards immediate danger is fair. This is especially true of syphilitic œdema, as it is more amenable to constitutional treatment. The "angioneurotic œdema" (urticaria of the larynx), as described by Quincke, the author, and others, is of transient duration and generally ends in recovery. If, however, the laryngeal œdema is due to nephritis, or cirrhosis of the liver, the ultimate prognosis is bad. About 50 per cent. of the cases of all types prove fatal. This mortality is not so remarkably high when we remember that most cases are due to renal disease.

Œdema of the larynx—treatment: If two facts are borne in mind, the indications for treatment become quite apparent. The two facts are (*a*) the local condition is one of serous infiltration in the loose areolar tissue of the larynx and epiglottis; (*b*) the œdema is usually due to some general disease which may produce œdema in other parts of the body.

The first fact suggests the propriety of puncturing the œdematous masses in order to relieve the intense tumefaction which causes respiratory obstruction. The Tobold concealed laryngeal lancet or other curved lancet may be used for this purpose. Repeat the scarifications every three to five hours.

The second fact suggests the propriety of treating the general disease causing the local laryngeal œdema. If cardiac disease is present, digitalis, strychnine, or strophanthus should be given. If nephritis or cirrhosis of the liver is present, elaterium or croton-oil should be given for its cholagogue effects. Pilocarpine, gr. $\frac{1}{8}$, should be given hypodermically at the same time for its diaphoretic properties. The heart's action should be closely watched, as pilocarpine is a cardiac depressant; if depression occurs, alcoholic stimulants should be promptly administered. Intubation, catheterization, or tracheotomy should be performed if cyanosis occurs. The syphilitic and tubercular types rarely advance so far as to require surgical interference.

Pachyderma of the Larynx.

Definition and pathology: This is a form of *chronic laryngitis* characterized by the formation of pavement epithelium on the free borders of the cords and in the interarytenoid space. The thickening of the epithelium is progressive; hence the peculiar "tumor" formations are developed. The subepithelial connective tissue is also thickened. The growths are usually on the cords along the posterior third,—*i. e.*, that portion of the cords corresponding to the arytenoid cartilages. The next most common location is in the interarytenoid space posteriorly. They are also occasionally found at the junction of the anterior and middle thirds of the cords. When located in this region they are known as *singers' nodules*.

The **symptoms** are those of chronic laryngitis associated with those of dry laryngitis. When the thickenings are on the posterior third of the cords one of them fits into a corresponding facet on the opposite tumor. On this account they do not interfere with the voice as much as would be expected from so large a lesion.

Prognosis: These thickenings do not affect longevity. If they are located on the posterior portions of the cords, the voice will probably remain somewhat impaired, especially for singing. If they are on the anterior two-thirds of the cords, they can be made to disappear so as to leave the voice unimpaired.

Pachyderma of the larynx—treatment: The catarrhal condition of the nose and pharynx should be attended to. Potassium iodide in four-grain doses should be given three times daily to promote secretion in the upper respiratory tract. *Locally* a spray of normal salt solution, or of a 2 to 3 per cent. solution of acetic acid, is of value. In some cases the acetic-acid solution will be too irritating. The perchloride of iron, 2 drachms to the ounce, or nitrate of silver, 1 drachm to the ounce, may be of some value in reducing the growths and relieving the symptoms. If, however, the "tumors" are large, they should be removed by surgical interference. As singers' nodules are due to the improper use of the vocal apparatus, they may be permanently cured by vocal exercises suited to the case.

Singers' nodules on the vocal cords: Reference has just been made to this condition in the preceding section. These nodules occur as the result of the improper use of the voice, and especially from exercises in which the cords are approximated, as in the production of the syllable AH. This exercise is known as the "stroke of the glottis." Its proper use gives brilliancy to the voice, while its improper use impairs it in the middle register. The cords are close together and nearly parallel when singing in this register, hence the nodules interfere with their proper vibration. Singing exercises which avoid the close approximation of the anterior two-thirds of the cords, as the use of the vowel O or broad "a" *preceded by* the consonant "m," as "ma," will, after two to four weeks, cause the nodules to disappear. Further information upon this point may be gained from "Voice-building and Tone-placing," by Holbrook Curtis.

Abscess of the Larynx.

Treatment: It should be opened with Tobold's curved laryngeal lancet (Fig. 141), the patient immediately throwing the head forward and downward to prevent the pus entering the trachea. If this precaution is not observed, suffocation may ensue. After a few moments of embarrassed respiration the desired relief follows.

Hyperæmia of the Larynx (Non-inflammatory).

Etiology; Hyperæmia of the mucosa of the larynx is due to any slight irritation or obstruction, whereby the arterioles are maintained in a dilated condition. Among these causes may be mentioned professional use of the voice, as in public speakers, criers, and singers, the inhalation of dust-laden air, and the excessive use of tobacco and alcohol. It may also be due to obstruction to the portal circulation; and cardiac and renal disease. Portal obstruction and cardiac insufficiency cause passive congestion, not only in the laryngeal mucosa, but in all the membranes of the body. Passive congestion is characterized by a bluish or purple color quite different from ordinary hyperæmia. The whole mucous membrane above and below the larynx is usually affected. Slight hypertrophy is sometimes present, especially after the condition has persisted for many months. The voice is uneven and cannot be depended upon for prolonged effort. Singers are especially annoyed by this impairment. There is an inclination to clear the throat of the cobwebby sensation produced by the hyperæmia.

Hyperæmia of the larynx—treatment: The treatment cannot be specifically given, as it varies with the causes. These should be carefully sought for and removed. The use of tobacco and alcohol should be strictly forbidden, and unusual use of the voice prohibited. The portal circulation should be regulated by the daily administration of a fresh infusion of senna leaves at bedtime. This should be prepared by taking a teaspoonful of leaves in two cups of cold water, which should be brought to the boiling-point, and then steeped for ten minutes. A cupful should be poured off and taken. It is of great importance that the tea should be freshly prepared each evening, as the resinoids are not held in solution after the infusion cools. It is of value in all diseases complicated by a gouty or rheumatic diathesis.

Anæmia of the Larynx in Incipient Phthisis.

The existence of pulmonary tuberculosis should be strongly suspected when laryngeal anæmia is present. The appearance of the membrane is ashen in color, as though a grayish film had formed over its surface. Fugaceous flushing of the membrane adds to the significance of the anæmia.

Tuberculosis of the Larynx.

Etiology: The *predisposing causes* are the same as those of tuberculosis in other organs of the body.

Catarrhal disease of the larynx probably favors its local manifestation, although this view is disputed by some writers. It occurs in the proportion of three males to two females. Those using the voice excessively seem to be more subject to it ; at least they lose their voices more quickly than those using the voice less violently. Tubercular laryngitis accompanies from 25 to 50 per cent. of the cases of pulmonary tuberculosis. It rarely coexists with tuberculosis elsewhere in the body. It may be primary in the larynx. It is most frequent between the ages of twenty-one and thirty years. The infiltration most often takes place in the interarytenoid space, spreading gradually to the arytenoid cartilages and superimposed mucosa, ventricular bands, and epiglottis. It may be limited to one side and later spread to the opposite side. It is usually bilateral.

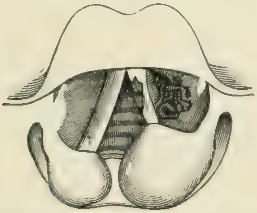
Tuberculosis of the larynx—symptoms: *Functional:* The loss of the voice is an early and frequent symptom. It sometimes changes suddenly from a gruff hoarseness to a high falsetto and thence into a toneless whisper. Dyspnœa may occur in advanced cases on account of the great amount of infiltration. There may be cough and pain followed by extreme exhaustion. Tubercle bacilli are usually found in the sputum, but their absence does not necessarily prove the case to be non-tubercular. Deglutition is sometimes impaired and attended by pain. The larynx is tender to the touch in advanced cases. Laryngeal hemorrhage is rare. The œdema if present is moderate, and in nowise forms a serious complication.

Physical: The mucosa in the early stages is a dull grayish-yellow or ashen colored. The cords may be red and the vessels of the mucosa injected. After a few weeks small yellow spots appear on the ashen-colored membrane ; these are the tubercles. The arytenoid cartilages and the mucosa covering them become infiltrated (Fig. 142) and club-shaped. When in this condition they are usually referred to as pyriform swellings. After a time they break down or ulcerate, and are covered with a thick, tenacious, ropy secretion slightly admixed with pus. The arytenoid cartilages become ankylosed and vocalization is still further interfered with.

Prognosis: The course of the disease is slow ; but inasmuch as it is almost always associated with pulmonary phthisis, finally leads to a fatal termination. In the meantime the voice becomes hoarse or aphonic, and in some cases deglutition becomes both painful and difficult.

Tuberculosis of the larynx—treatment: This should be as for pulmonary tuberculosis with the addition of such local measures as give comfort, and perhaps, in some instances, prolong life. In primary tuberculosis of the larynx the complete removal of the infiltrated areas by curettement is, sometimes, followed by permanent cure. The condition is, however, almost always secondary, thus rendering a cure much less probable. The results are nevertheless so favorable that active treatment is to be recommended. The treatment depends upon the stage and location of the process. If it is in the pre-ulcerative stage, simple alkaline washes or sprays should be used locally, so as to remove the secretion and stimulate regenerative processes, thereby prolonging this stage and postponing the appearance of the stage of ulceration.

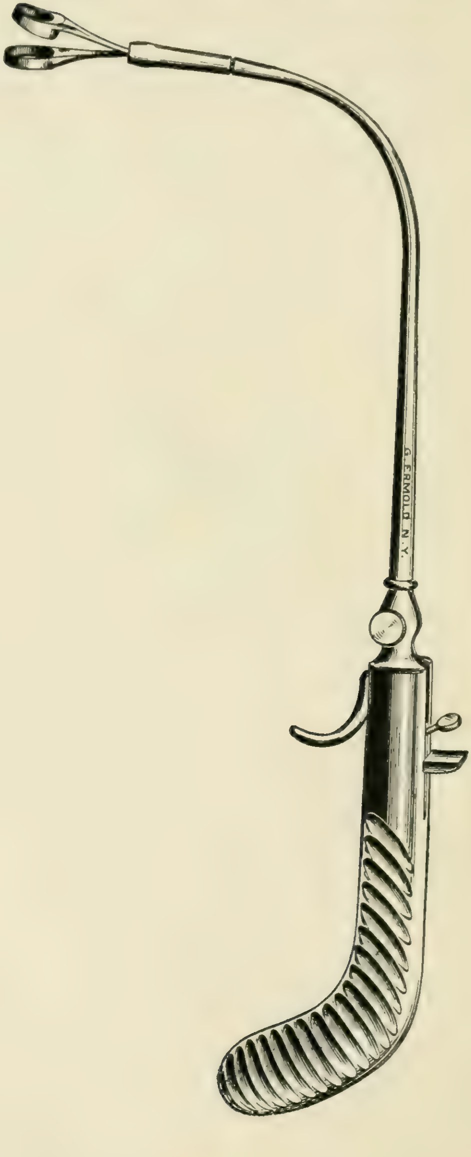
FIG. 142.



A tubercular ulcer on the left ventricular band and left vocal cord. Pear-shaped œdematous swelling of aryepiglottic folds, more intense on the side of the ulceration. (Cohen.)

Most cases are not seen until the *stage of ulceration* is well developed; hence this condition is usually the one presented for treatment. An effort should be made to excite mild reactionary regeneration of the ulcerated surface. This can be done by first cleansing the parts with alkaline washes, followed by the use of a spray of hydrozone. The parts should then be dried with a cotton-wound applicator and painted with dilute hydrochloric acid or with a 50 per cent. solution of lactic acid. The ulcers are sometimes very deep and exten-

Fig. 143.



Coakley's double curette for removing intralaryngeal growths.

sive, and do not respond to this mode of treatment. Such cases should be curetted deeply with Coakley's (Fig. 143) or other laryngeal curette, care being taken to remove all of the tuberculous tissue. The limiting membrane which surrounds the diseased area will be broken, and if all tubercular tissue is not removed it may extend to the adjacent tissues. Mere curettement is worse than useless, but the thorough removal of the diseased tissue may be of the greatest value. The application of acids after curettement is of value chiefly as a destructive agent to the remaining fragments of tubercular tissue, and it also excites phagocytosis, which raises the vital resistance of the parts.

Pain is a most distressing symptom in many cases, especially upon swallowing. In such cases liquid foods should be given and such anodyne remedies as afford the greatest amount of relief. For this purpose cocaine sprays or powders containing 10 per cent. of cocaine should be applied to the laryngeal mucosa. In very aggravated cases morphine may be used locally and by hypodermic injection. The use of pineapple juice with a spray or applicator has been attended with marked success in relieving pain. The vegetable astringents, as hydrastis canadensis, combined with ergot, administered internally, afford relief to the cough. They should be prescribed as follows:

℞. Fluid extract canadensis,
  Fluid extract ergot,   āā f℥ iv.—M.
Sig. Take 10 to 40 drops four times a day.

For the relief of the dryness there is nothing better than the comp. wine of iodine given in teaspoonful doses with three or four ounces of water. Locally, oily sprays containing 10 per cent. of menthol add to the comfort of the patient, and are extolled by some as a remedy of great value. I have seen no benefit from the use of electricity in any form.

The use of guaiacol and creosote internally pushed to the point of toleration, combined with the administration of cod-liver-oil, the hypophosphites, iron, and arsenic, often exerts a marked controlling effect upon the progress of the disease. In cases not far advanced climatic treatment should be advised after having thoroughly removed the local morbid condition with the curette and acid applications. The patient should not be sent away unless he is placed in charge of a competent local physician; or is provided with the means of keeping a clinical record of his case, which is mailed to his physician at stated intervals.

Scleroma of the Larynx.

Etiology: While but few cases have been reported in this country, in Germany many are found. It is due to a specific microörganism, and is secondary to rhinoscleroma.

Pathology: The mucous membrane is pale and gristly and extensively infiltrated. In the course of the disease *ulceration* occurs. The ulcers are shallow nodular erosions covered with dried pus and inflammatory *débris*. The epiglottic mucosa is more especially liable to be involved, and the scabs may be seen through the chink of the glottis.

The **diagnosis** may be rendered certain by examination for the characteristic germ in the tissue.

The **treatment** should be expectant, as no remedy of value is known. Should stenosis occur, tracheotomy affords a means of relief.

Pemphigus of the Larynx.

This is a rare condition, *characterized* by an herpetic eruption of the larynx, usually limited to the ventricular bands.

There is a slight constitutional disturbance, with local sensation of heat. The condition is often attended by gastro-intestinal disorder, the regulation of which should form a prominent part of the *treatment*. The mouth and pharynx should be frequently washed with an antiseptic gargle.

Syphilis of the Larynx.

As the **initial lesion** is rarely, if ever, found in the larynx, consideration will be limited to the secondary and tertiary stages.

Syphilis of the Larynx—Secondary Lesions.

Secondary syphilitic manifestations are *erythema*, the *mucous patch*, the *superficial ulcer*, and *condylomata*.

Erythema of the laryngeal mucosa is not usually coincident with the skin eruption, but follows it after a period varying from a few weeks to six months. The administration of the iodide of potassium causes its rapid disappearance.

The superficial ulcer is irregularly oval, extending its borders slowly. One or more ulcers may form at the same time, or may appear after the disappearance of the preceding ulcer. On account of this peculiarity it has sometimes been called "recurrent ulcerative laryngitis." After the subsidence of the ulcerative stage a superficial stellate scar is formed. The superficial ulcer occurs most often between the second and seventh years after infection, and yields readily to the classical treatment.

The **mucous patch** is rare, and according to some authors never appears upon the larynx. Those who have described it say that it always appears above the glottis.

Condylomata appear as yellowish pimples with elevated bases. They soon break down and discharge their contents, causing little or no discomfort or functional disturbance.

Syphilis of the Larynx—Tertiary Lesions.

The changes characteristic of this stage rarely take place before the fifth year, but usually between the tenth and twenty-fifth years, although they may occur at a much later period

The pathological phenomena are *gummy tumor, deep ulcer,* and *cicatricial stellate scars.*

The **gummy tumor** is a small round-celled infiltration which may be either diffused or circumscribed.

The *diffused infiltration* involves the submucosa, muscles, and perichondrium; hence the movements of the larynx are interfered with. The infiltration occurs slowly, the membrane being red and sometimes œdematous. The œdema is a serous infiltration of the submucous tissue, and may obscure the true nature of the disease. After a time the diffused infiltration may undergo degenerative changes and become irregular superficial ulcers.

The infiltration may be *circumscribed* and stand out from the surface of the mucosa as a clearly defined tumor, or true gumma. The circumscribed gummy tumor persists but a short time, as it usually undergoes rapid degeneration. The breaking-down process begins in the centre of the new formation in the form of fatty degeneration, which rapidly spreads to the periphery of the growth, thus merging into the ulcerative stage.

The **deep ulceration** which results has clear-cut, punched-out edges with perpendicular walls and a greenish-gray bottom. The ulcers throw off a foul-smelling semi-fluid discharge. One portion of the membrane may be in the ulcerative stage, while other portions are still in the stage of infiltration or gummatous swelling. The ulcers often form upon the upper surface of the epiglottis, and on the true cords, as these are the most exposed portions of the larynx.

Postulcerative or cicatricial scars form in the parts previously destroyed in the ulcerative process. The cicatricial bands usually radiate from the centre of the old ulcer, thus forming the peculiar **stellate scars** which are so characteristic of syphilis of the mucous membrane. The radiating bands show a marked tendency to contraction, and in consequnce the tissue about them becomes much distorted. Stenosis of the larynx may occur, and suffocative symptoms intervene, which may require speedy tracheotomy to prevent a fatal termination. Mixed infection may remain long after the syphilitic inflammation has entirely subsided, and may require special treatment for its removal.

Syphilis of the Larynx—Symptoms.

The symptoms of the **period of infiltration** extend over several weeks or months, as it progresses very slowly. There are slight discomfort and pain upon swallowing, and a tickling or scratching sensation in the throat. The voice is more or less hoarse or aphonic according to the degree and location of the infiltration. If it is of the circumscribed sort, the motility of the vocal cords will be impaired. If the true cords are involved, there will be partial or complete aphonia. Strangulation upon attempts to swallow fluids may occur in extensive infiltration of the epiglottis.

In the **stage of ulceration** the pains become more marked, and are stabbing and fiery in character, especially after taxing the voice or upon awakening in the morning. The movements of the epiglottis may be circumscribed on account of the infiltration of its substance, and of the muscles controlling it; hence dysphagia may become a distressing symptom.

The symptoms of the **cicatricial stage** are those due to interference with vocalization, respiration, and deglutition.

Syphilis of the larynx—Diagnosis.

It may be confounded with tuberculosis and malignant diseases of the larynx. The general symptoms and history of the case, when considered in connection with the local phenomena, generally afford a ready means of diagnosis.

In **tuberculosis** the presence of tubercle bacilli in the sputum, the pulmonary signs, and the local laryngeal anæmia form a symptom-complex, which should not be mistaken for syphilis.

Malignant disease of the larynx is more apt to be characterized by paralysis of one or both vocal cords, and by a cachectic appearance, which, in the absence of a syphilitic history, renders the diagnosis comparatively easy.

Syphilis of the Larynx—Treatment.

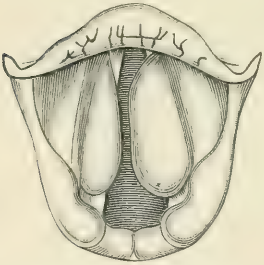
The treatment to be curative must be given in the stage of infiltration. After the parts have been destroyed by the ulcerative process it is too late to prevent the serious consequences of the disease. Potassium iodide should be given, in combination with the fluid extract of gentian or the carbonate of sodium, in doses as large as the patient will tolerate. I find it of advantage to administer the iodide in several ounces of hot water, or in milk, sometimes giving as much as one hundred and twenty-five grains three times daily after meals. It is best administered in a saturated solution, beginning with ten or fifteen drops after meals, increasing the dose one drop daily until the point of toleration is reached. If iodism or stomach irritation occurs, the dose should be diminished one-half and again gradually increased to the point of toleration. Mercurial inunctions or hypodermic injections of the bichloride of mercury should be given with the iodide treatment.

Should severe dyspnœa occur from œdema, infiltration, or stenosis, appropriate surgical treatment should be undertaken to relieve the symptom. Sequestra of the laryngeal cartilages should be removed with curved laryngeal forceps. Deep abscesses should be evacuated externally or internally according to the indications.

Prolapse of the Laryngeal Ventricles; Eversion of the Ventricles.

Etiology: During syphilitic or tubercular laryngitis a violent attack of coughing may loosen the mucous folds from their attachments to the parts beneath, and allow them to protrude above the larynx or over the true cords (Fig. 144).

Fig. 144.



Prolapse of the ventricles of the larynx. (Coakley.)

The **symptoms** are those due to the interference with the respiratory functions, and the movements of the vocal cords.

Treatment: Painting the parts with astringent solutions will relieve the symptoms in so far as they are due to reactionary inflammation, but will have little influence in overcoming the obstruction caused by the misplaced mucous membrane. The ablation of the dislocated ventricular bands with a snare or laryngeal forceps through the mouth, or after thyrotomy, affords the best means of treatment at our command.

Lupus of the Larynx.

Pathology: Lupus of the larynx is a tubercular infection which is limited to the superficial layers of the mucous membrane. Numerous elevated masses appear on the infected areas and there is a marked reactive proliferation of epithelium. Ulcerations occur sooner or later. The progress of the disease is uncommonly slow, both before and after the ulcerative process is established. Lupus of the larynx, or other portions of the respiratory tract, is almost always *secondary to lupus of the skin.* The process in the respiratory tract begins in the nose and passes downward to the pharynx and larynx. It may, however, be primary in the latter.

It **occurs** most often in people residing in the country, and in the female sex. It is a disease of middle life, although all ages may be affected.

It is not hereditary, but the lymphatic or strumous diathesis **predisposes** to the infection. A *slight local traumatism* may excite an acute exacerbation of the pathological process.

Lupus of the larynx—symptoms: Deglutition is sometimes attended with pain, while the voice is more or less impaired according to the degree and location of the diseased areas. If the true cord is the seat of lupous inflammation, there will be hoarseness or complete aphonia. Dyspnœa is present in a few cases in which there is a pronounced tumefaction of the tissues. This rarely occurs, however, as the process is too slowly destructive for the production of œdema. Reconstructive infiltration does not figure permanently in this disease, and yet there is but little destruction of tissue, as the process is limited to the superficial layers of the mucosa, and the destructive process is very slow. Pain is usually absent, and any part of the larynx may be affected. Cough may or may not be present.

The **general health** is usually good.

Laryngoscopic examination reveals a nodular ulcer, with a pinkish-gray bottom, slightly below the surface of the mucous membrane. There is little or no œdema, the mucous membrane being slightly hyperæmic.

Lupus of the larynx—treatment: Aqueous solutions of the nitrate of silver, containing from 120 to 480 grains to the ounce, applied locally by means of a laryngeal brush or cotton-wound applicator often exert a very favorable influence on the course of the disease. The topical application of the tincture of iodine, the galvanocautery or fused chromic acid crystals have been used with more or less success. The lymphatic diathesis should be corrected by the administration of codliver-oil, iron, arsenic, and strychnine; and by placing the patient in suitable hygienic environment. Curettement of the ulcers, followed by the application of a 50 per cent. lactic acid solution, does not seem to act as favorably as it does in true tuberculosis. Excision of the lupous mass, including some of the surrounding healthy tissue, is sometimes followed by improvement.

Leprosy of the Larynx (Elephantiasis Græcorum; Lepra).

Leprosy of the larynx is never primary, but is one of the attendant phenomena of general leprosy.

It is **characterized** by hard nodular infiltration, ulcers, and cicatricial contraction which is apt to produce stenosis. The stenosis is, however, in part due to the infiltration, which, when excessive, involves the lumen of the larynx. Extensive destruction of tissue often occurs in which the epiglottis is most apt to be involved.

The disease is **due** to the bacillus lepræ, and is most common in China, India, and the Hawaiian Islands; and between the ages of fifteen and thirty years. It is slightly contagious, and probably hereditary.

Gouty Inflammation of the Larynx.

This is a rare condition, and is *characterized* by gouty deposits upon the epiglottis and laryngeal cartilages. These offer a hard and gritty sensation to the sense of touch when probed. The membrane is swollen and yellowish-red in circumscribed areas. These symptoms, when taken with the general history of gout, render the *diagnosis* comparatively easy. The *treatment* should be addressed to the relief of the gouty diathesis.

Arthritis Deformans of the Larynx.

Here, as elsewhere, the articular surfaces are the seat of inflammatory deformity. Motion of the vocal apparatus is interfered with, or the ankylosis may be complete.

FOREIGN BODIES (in the Larynx, Trachea, and Bronchi).

Occurrence: Any solid, fluid, animate or inanimate object may gain entrance into the lower air-tract from without by way of the mouth or nose; or from within by the rupture of an abscess of the pharynx, larynx, or other adjacent tissue; and from exfoliation of the laryngeal cartilages.

The **symptoms** vary somewhat with the size, location, and physical character of the object.

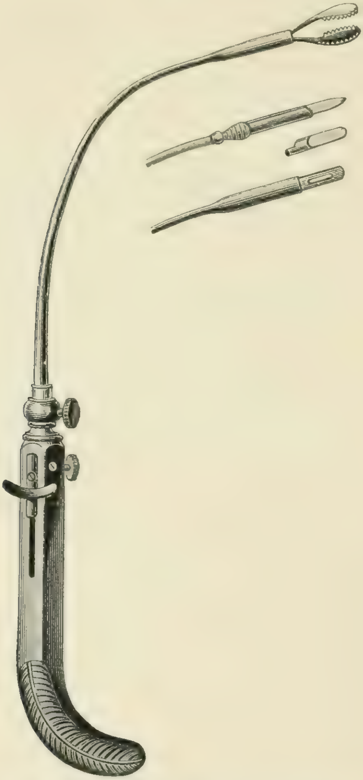
The *entrance* of the foreign body into the air-tract is signalized by a sudden choking, gasping inspiration, and marked dyspnœa. There is an anxious expression of the countenance, the eyes being wide open and protruding, while the patient seeks wildly for an open door or window where he may obtain plenty of fresh air. Suffocation may take place and necessitate an immediate tracheotomy to prevent a fatal termination.

If the foreign body *passes into the lower tract*, the dyspnœic symptoms subside or entirely disappear, only to recur at varying intervals. The foreign body may be tolerated for an indefinite length of time, one case being reported in which the foreign body was expelled after having been in the trachea for sixty years.

After a time inflammatory and ulcerative processes may develop in the parts holding the foreign body. Hoarseness and aphonia, and recurrent attacks of dyspnœa mark the later history of retained foreign bodies. The expectoration may be streaked with blood. A slight febrile movement, attended by a hectic flush of the cheeks, closely simulating pulmonary phthisis, may occur in the progress of the reaction-inflammation. The foreign body is most apt to enter the right bronchus, as it is in a more direct line with the trachea.

Foreign bodies—treatment: If suffocative symptoms persist, tracheotomy or laryngotomy should be performed. They usually subside, however, before such extreme measures become necessary. Effort should then be made to locate the foreign body. The patient should be placed upon his back in a recumbent posture, with his head hanging down over the end of the table, so that gravity will not tend to carry the foreign body still further into the respiratory tract. This position also favors the expulsion of the offending mass, as the posterior portion of the glottis does not offer marked interference to its exit through the chink in the glottis. The anterior portion of the glottis, however, offers considerable obstruction to the exit of the body. While in this position laryngoscopic and digital examination of the larynx should be made. The trachea should also be examined with the laryn-

FIG. 145.



Tuerck handle, containing serrated forceps. Two sheathed knives and a sheathed caustic applicator are also shown.

goscopic mirror to ascertain if the foreign body is above the bifurcation. The chest should also be carefully explored by auscultation and percussion, to determine, if possible, the exact location of the foreign body.

Sometimes the inversion of the patient is attended by the immediate expulsion of the foreign body. While the patient is thus inverted, he should be smartly struck across the chest with each respiratory movement, in order to facilitate the dislodgment of the body. If it is located in the chink of the glottis or immediately below it, it may be seized with a curved laryngeal forceps (Fig. 145) and removed. Should it be lodged low down in the trachea, tracheotomy should be performed and the severed cartilaginous rings spread wide apart, when the body is often voluntarily expelled. If not, it can be removed, through the artificial opening, with slender curved forceps. Forced inflation through the tracheal opening is sometimes followed by its expulsion. Titillation of the interior of the trachea with a feather may be attended by a like favorable result. A tracheotomy-tube should not be inserted through the opening, as it interferes with the expulsion of the object. The tracheal opening should be spread apart with strong threads passed through the severed rings and passed back of the neck and tied. If the foreign body is not removed, or its location definitely determined, the tracheal opening should be maintained for further exploration. It is important that the temperature of the room should be maintained at about 75° F., and the atmosphere constantly charged with moisture from boiling water. Failure to observe these precautions may result in irritation to the lower air-tract and thus favor the development of pneumonia.

To remove the foreign body from the bronchus the posterior mediastinal operation may be performed.

NEUROSES OF THE LARYNX.

Sensory Neuroses.

Anæsthesia of the larynx may include insensibility to *touch*, *heat*, and *cold*, either separately or together. It may be unilateral or bilateral, and above or below the glottis, according to the seat and nature of the lesion causing it.

Etiology: It may be due to hysteria, or to a neuritis following diphtheria, syphilis, or meningitis; or to pressure on the trunk of the recurrent laryngeal. It is present in general paralysis of the insane, in tumors at the base of the brain, and in the early stages of tabes dorsalis, or other diseases affecting the medulla.

Treatment: If the anæsthesia is peripheral in origin, the faradic or combined faradic and galvanic currents should be applied locally by means of suitable electrodes, the indifferent electrode being placed on some remote part of the body. Strychnine is a valuable adjunct to the electrical treatment. Iron, phosphorus, and arsenic are indicated in neurasthenics. If due to tumors or other organic diseases of the brain, treatment is useless.

Hyperæsthesia of the larynx: It is a common symptom of acute simple inflammations of the larynx, tubercular laryngitis, and sometimes of chronic laryngitis.

It is present in neurasthenia, anæmia, prolonged dyspepsia, and in chronic alcoholism. There may be transitory hyperæsthesia of the larynx during teething, menstruation, and pregnancy. It may be the first sign of phthisis, hence often furnishes a valuable aid in the early diagnosis of this important disease.

Increased reflex irritability is the chief *symptom* of laryngeal hyperæsthesia. The *prognosis* depends on the cause underlying it. If it is neurasthenia, disordered menstruation, or pregnancy, it is favorable; if due to tuberculosis, it is unfavorable.

The *treatment* should be addressed to the cause, rather than to the hypersensitiveness itself.

Paræsthesia of the larynx: This is a perverted sensation in the larynx, the patient complaining of a tickling or prickling in the throat, or of the presence of a foreign body. As suggested by the title, the condition is not a real one, but a perverted sensation neurosis. These sensations are to be accounted for by obstructed venous circulation and hypertrophy or other diseased states of the lingual and faucial tonsils. In other cases they are due to slight tissue-changes, as in incipient tuberculosis.

Neuralgia of the larynx: The usual signs of neuralgia are present, as pain and tender spots, the pain being either constant or paroxysmal. It may be spontaneous or caused by attempted phonation, as in hysterical individuals. Phosphorus, arsenic, quinine, iron, and strychnine should be administered internally, and the galvanic current applied locally. The positive electrode of the battery should be applied through the mouth to the laryngeal mucosa, while the negative pole should be placed on some remote part of the body. Should the poles of the battery be reversed in their application, the pain will be increased rather than decreased. The dose of the current should be limited to about 5 ma., as more than this is apt to excite inflammatory reaction, or produce actual destruction of tissue by electrolysis.

Hyperkinetic Neuroses (Spasmodic Neuroses of the Larynx).

The spasms may be either *tonic* or *clonic*.

Tonic Spasms of the Laryngeal Muscles.

Their origin may be (*a*) *central;* (*b*) from *irritation* of the *trunk* of the *recurrent laryngeal;* and (*c*) from *reflex irritation.*

Tonic spasms of central origin: In tabes dorsalis spasm of the adductors of the larynx occurs. *Clinical picture:* Sudden dyspnœa with loud inspirations, the cords remaining in adduction for some time. It also occurs in tetanus, tetany, and hydrophobia.

Tonic spasm from irritation to the trunk of the recurrent laryngeal: When the injury is transient and slight there will be a laryngeal spasm which is a forerunner of paralysis. Aneurism of the arch of the aorta, cancer of the œsophagus, pleuritic adhesion of the right apex of the lung, and tumors of the mediastinal glands may cause the irritation. Slight lesion may also occur in tabes.

Tonic spasms from reflex irritation: These may occur from irritation to the larynx, fauces, and neighboring parts. In highly sensitive children, irritation in a remote part of the body may cause adduction spasms. The latter condition has been described as *laryngospasmus infantum*, and is usually due to intestinal irritation, tapeworms, a tight prepuce, or constipation.

Clonic Spasms of the Laryngeal Muscles.

These are always of **central origin.**

They consist of rhythmical inward movements of the cords. The condition may last but a few moments, or may persist for many months. The pillars of the fauces are also often affected in a like manner.

The *tonic* and *clonic* spasms may be present in the same case, especially in the depressors of the epiglottis. The diseases most often *causing* clonic spasm of the larynx are syphilis, meningitis, and intracranial tumors.

Treatment of Laryngeal Spasms.

Of central origin: It should be *symptomatic*, as the diseases causing it are incurable. Prolonged *tonic* spasms of the adductors, resulting in marked dyspnœa, should be relieved by tracheotomy. Morphine injections will reduce excessive excitability.

Of reflex spasms: These are usually seen in children, and should be arrested, as more or less danger attends them. The child should be placed in a warm bath and the posterior part of the chest douched with cold water. The intestinal tract, portal circulation, and genitalia should receive careful attention and, if necessary, appropriate treatment.

Of spasms from irritation of the recurrent: Little can be done for spasms from irritation of the recurrent laryngeal nerve, as it is usually due to intrathoracic pressure from some grave tumor or other morbid condition.

Laryngeal Vertigo (Laryngeal Apoplexy; Laryngeal Syncope; and complete Glottic Spasm in Adult).

It is **characterized** by transient irritation in the throat, followed by a fit of coughing, dimness of vision, dizziness and unconsciousness, the patient falling to the floor.

Symptoms: The face is flushed. There may be epileptiform convulsions, which, after a little while, are followed by a return of consciousness. After such an attack all signs of the condition disappear. The seizure is clinically much like apoplexy with laryngeal aura and laryngeal spasm, which is continued long enough to produce unconsciousness. Such spasms are liable to occur in neurasthenics and in tabes. Other signs and suggestive family history, which characterize epilepsy, tabes, and neurasthenia should be sought for before pronouncing the case one of laryngeal vertigo.

The **treatment** should be addressed to the correction of alimentary and portal disorders; and to the excretory organs of the body. Tonics, arsenic, and phosphorus are sometimes of value in preventing a return of the laryngeal disorder.

Nervous Cough.

Definition: This is a spasmodic, croupy, or even musical, laryngeal cough, for which no physical cause can be assigned.

Symptoms: Persons thus affected are neuropaths presenting other stigmata of neurosis. The cough is usually persistent during the daytime, being increased upon excitement, and subsiding during sleep. Upon awakening in the morning it returns with renewed vigor.

Treatment: Ten-grain doses of the bromide of potassium or sodium should be given internally three times a day. The larynx should be sprayed with aromatic oily solutions of menthol and eucalyptol. Systemic tonics, laxatives, outdoor exercise, together with occupancy of a room with abundant exposure to sunlight, should be made a part of the daily regimé.

Mogiphonia.

This is **characterized** by difficulty in maintaining the tension of the vocal cords while singing or during forced accentuated speaking. In ordinary conversation no difficulty is experienced.

Rest, massage, friction, and regulation of alimentary and portal disturbances afford the best means of **treatment**.

Chorea of the Larynx (Laryngeal Nystagmus).

This condition is quite like chorea in other parts of the body, and is **characterized** by frequent recurrences of a sharp, dry, noisy cough resembling a bark or yelp of a dog. Here, as in chorea of the arms and face, the disease is usually manifested in females about the age of puberty.

Symptoms: The yelp or bark consists of a sudden expiratory effort, or of a series of similar noises gradually diminishing in intensity. Choreic movements are usually, though they may not be, present in other parts of the body. The voice is unaffected, but may betray a slight jerkiness in articulation.

The patient should be **treated** as for general chorea, with the addition of sedative inhalations of a hot vapor of the compound tincture of benzoin; or of the vapor from a hot infusion of hops or hyoscyamus. Bromides and arsenic should be administered internally, the bowels and portal circulation regulated, and the activity of the skin established by friction and cold douches.

Hypokenitic Neuroses (Laryngeal Paresis and Paralysis).

Motor innervation of the laryngeal muscles is by the superior and recurrent laryngeal nerves. The *recurrent* is most frequently involved.

Total Unilateral Paralysis of the Recurrent.

The **clinical picture** is as follows:

a. The *affected cord* is fixed midway between the respiratory and phonatory positions,—*i. e.*, in the cadaveric position. Neither the abductors nor the adductors are acting. The cord is in a state of passive equilibration.

b. The *free edge* of the affected cord is more concave than normal.

c. The *arytenoid cartilage* on the *affected side* is rotated somewhat inward, thus allowing the vocal process to project into the chink of the glottis, and convert the concave edge of the cord into two concave segments.

d. The *arytenoid cartilage* on the *unaffected side* moves forward during phonation somewhat in advance of the one on the affected side.

e. *During phonation* the paralyzed cord and the arytenoid cartilage remain fixed; while those upon the unaffected side move inward somewhat beyond the median line. The exaggerated inward movement is a compensatory effort at vocalization.

f. The *glottic opening* is oblique from before backward toward the affected side.

g. The *arytenoid muscle* on the *affected side* sometimes twitches, as it is occasionally supplied by the superior laryngeal, which is not usually involved. This sign should enable the observer to exclude ankylosis and prove the nervous origin of the impairment.

h. The *epiglottis* is usually innervated by the superior laryngeal, but is occasionally supplied by the recurrent. When by the latter the epiglottis may be seen to twitch upon the sound side.

i. The *voice* is at first feeble, slightly roughened, but not hoarse. Later on, these conditions disappear and the voice becomes nearly natural. This is due to compensatory function on the unaffected side.

j. There is excessive *waste of air* during phonation, and the respiratory act is necessary after speaking a few words.

Total Bilateral Paralysis of the Recurrent.

The **clinical picture** is as follows:

a. There is *complete aphonia,* as both cords are in the cadaveric position.

b. There is great *waste of air* upon attempted phonation.

c. There is *no motion of the cords,* as both the adductors and abductors are paralyzed.

Paralysis of the Abductors.

In this condition a portion of the fibres of the recurrent pharyngeal are involved, and the following **clinical picture** is presented:

a. There is almost *complete closure* of the glottis (Fig. 146), giving rise to

b. *Dyspnœa;* and

c. *Stridor.* Both are increased during sleep or upon slight irritation of the larynx.

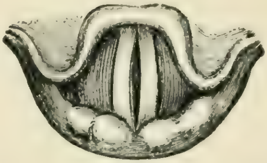
d. The dyspnœa and stridor are *greater during inspiration.*

e. The *voice* is usually unaffected in ordinary conversation.

f. The *cords* remain almost parallel, being more nearly approximated during inspiration. This sometimes results in profound dyspnœa.

Prognosis of recurrent laryngeal paralysis: In paralysis either of the muscles of abduction, adduction, or both combined, the prognosis is usually serious, as the conditions causing it are incurable or very grave. The dyspnœa may be so marked as to call for tracheotomy.

FIG. 146.



Position of the vocal cords in respiration in bilateral abductor paralysis. (Cohen.)

Laryngeal Paralysis from Various Causes.

It may result from *injury* to the *pneumogastric* by fracture at the base of the skull; or to the spinal accessory; or from bulbar disease.

Postdiphtheritic bulbar degeneration sometimes suddenly develops, and causes laryngeal paralysis. When disseminated sclerosis invades the medulla and involves the laryngeal centres the symptoms are bilateral. Tabes is the type of degeneration which most often impairs the laryngeal centres. In this disease the paralysis may be bilateral or unilateral, complete or incomplete.

Atrophy of the Vocal Cords.

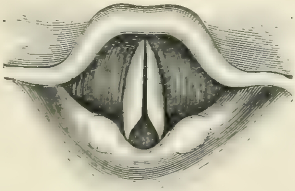
Atrophy may arise from any condition which prevents the use of the vocal organs, as paralysis of central or peripheral origin; and from inflammatory infiltration and degeneration of the muscular fibres themselves. It may also follow ankylosis of the arytenoid cartilages, or prolonged fixation of the cartilages from rheumatic laryngitis. Faradism may be applied locally in those cases which have a prospect of ultimate recovery.

Hysterical Paralysis (Functional Aphonia).

Etiology: Great mental or physical exhaustion resulting from sickness, prolonged anxiety, or physical exertion is sometimes attended by complete loss of the voice. It may be due to the inhibition of the will-centres brought about by fright or powerful suggestion. Incipient tuberculosis is sometimes ushered in by this type of aphonia. Occasionally chronic laryngeal catarrh seems to be the cause.

Hysterical paralysis — symptoms: The voice (voluntary muscular action) is lost, while coughing and laughing are phonetic, as they are involuntary acts. Speech, being voluntary, is inhibited. *Inspection* shows the cords separated, moving slightly inward upon attempted phonation, and slightly outward upon deep inspiration (Fig. 147). The arytenoideus muscle may alone be affected and cause hoarseness and fatigue upon phonation.

FIG. 147.



Paralysis of the arytenoideus. (Coakley.)

Hysterical paralysis—treatment: Tonics, nutritious food, plenty of exercise in the open air, sunshine, freedom from mental anxiety, and excessive physical exercise will do much toward effecting a cure. Suggestive therapeutics will aid greatly in overcoming the inhibition of the will, one of the most important etiologic factors of the condition.

TUMORS OF THE LARYNX.

Benign Tumors of the Larynx.

Classification: Papillomata, fibromata, cystomata, myxomata, adenomata, lipomata, angiomata, and enchondromata.

Etiology: Hyperæmia is regarded by some as a cause, while other observers of equal repute do not so regard it. The same is true of catarrhal inflammations.

Benign tumors are most common in middle life, but are occasionally found in the very young as well as in the old. They have been occasionally described as congenital. They are most common in males, probably on account of a more exposed life and pernicious habits. Overtaxing the voice, as in violent singing, street-crying, auctioneering, and public speaking, seems to be a prominent cause. The inhalation of irritating vapors, as from soapsuds-steam, etc., has more or less influence in their causation. Nasal obstruction probably exerts some influence; while syphilis and tuberculosis undoubtedly exert a marked influence in their causation.

Benign tumors of the larynx—symptoms: The voice is impaired in about nine-tenths of the cases, the impairment usually being in the form of hoarseness or aphonia. Occasionally, however, it may be double—*i. e.*, gives off two tones at once. This is caused by small growths which divide the cords into two segments, each segment giving forth a different tone. If there is no hoarseness or other impairment of the voice, its volume and strength are diminished. In about 15 per cent. of the cases there is marked dyspnœa. Cough may or may not be present. If present, it is probably due to the pressure of the growth upon the "cough-spots of Stoerck." The expectoration is scanty and sometimes streaked with blood. Deglutition is interfered with when the growth is upon the epiglottis, in the hyoid fossa, or upon the wall of the pharynx. There may be some discomfort, but no pain. In adults the mucosa is not usually inflamed. In children, however, the growths are usually attended by laryngitis, which gives rise to nocturnal dyspnœa.

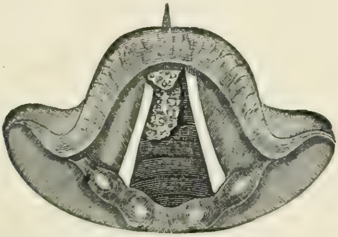
The *laryngoscopic image* varies with the location and character of the growth.

The cough-spots of Stoerck: The sensitive areas known as "the cough-spots of Stoerck" are located on the (*a*) posterior commissure between the arytenoid cartilages; upon the (*b*) posterior wall of the larynx and (*c*) trachea; and upon the (*d*) bifurcation of the trachea.

Benign Tumors of the Larynx—Diagnosis.

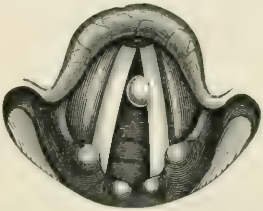
Papillomata: They are soft, pinkish-gray or whitish-gray, wart-like growths (Fig. 148). They are usually movable, and grow from the anterior angle of the cords. I have seen one case in which they grew from the whole length of the cords and from the under surface of the epiglottis.

Fig. 148.



A small papilloma springing from the anterior commissure and resting on the right vocal cord. A papilloma also is shown arising in the subglottic region of the larynx beneath the right vocal cord. (Cohen.)

Fig. 149.



Fibroma on the left vocal cord.

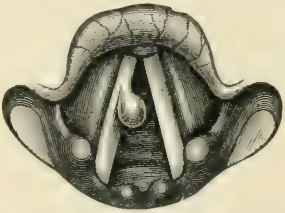
Fibromata: They are firm to the touch, have a rounded nodular contour (Fig. 149), and the mucous membrane covering them is somewhat injected. They are either sessile or imbedded in the surrounding tissues. They most often grow from the anterior portion of the cords. In general appearance they somewhat resemble cystomata and chondromata. Chondromata never grow from the cords, and are much firmer to the touch; while cystomata are soft, movable, and semitranslucent.

Cystomata: They pit upon probe-pressure and regain their rotundity at once. They are small, roundish, pinkish-gray, semitranslucent tumors. When lying across the chink of the glottis they appear almost transparent.

Chondromata: As chondromata grow from the cartilaginous box they are covered with healthy mucous membrane. They develop very slowly, the cricoid cartilage being most often affected. In this location they may resemble perichondritis or carcinoma. In *perichondritis* there is a history of sudden onset, followed by marked dyspnœa, which renders the differentiation clear. *Carcinoma* of the larynx rarely grows below the glottis, hence need not be confounded with chondroma of the cricoid.

Laryngeal polypus: This is a rounded semitranslucent tumor usually attached to the vocal cords, as shown in Fig. 150.

Fig. 150.



A polyp on the right vocal cord.

They are slightly pedunculated and more dense than polypi of the nasal mucous membrane.

Benign Tumors of the Larynx—Treatment.

This is chiefly **surgical**.

Nearly all benign tumors may be removed through the mouth with a snare, cautery, or one of the curved curettes shown in Figs. 139 and 143. If the growth is pedunculated, the snare loop may be insinuated around it; and if it is sessile, one of the laryngeal biting or cutting curettes should be used. Small growths upon the edge of the cords are best removed with the serrated laryngeal forceps shown in Fig. 145. Care should be exercised to avoid injury to the cords, as permanent impairment of the voice might follow.

Local anæsthesia should be produced by the application of a 20 per cent. solution of cocaine to the laryngeal mucous membrane, otherwise the spasmodic movements of the pharynx and larynx will interfere with the performance of this operation. Great dexterity from large experience is required for these operations. The operator does not see the growth, but an inverted image of it. The same is true of the cutting end of the laryngeal instrument. He is guided by the reverse movements observed in the laryngeal mirror. This makes the operation a very difficult one. The beginner should practise laryngoscopic examination and instrumentation upon a large number of persons before attempting to remove morbid tissue from the larynx.

Malignant Tumors of the Larynx.

Sarcoma.

Definition: Sarcoma of the larynx is an embryonic connective-tissue growth usually occurring between the twentieth and fortieth years. The larger celled tumors grow slowly; while the smaller celled ones develop rapidly. The tumor is rarely found in the larynx, being in the proportion of one to sixty-two of carcinoma. The **etiology** is obscure.

Sarcoma of the larynx—symptoms: Vocalization and respiration are impaired early. There is a spasmodic hacking cough, due to the presence of secretion. Cough is a distressing symptom when the growth is infraglottic. Dysphagia is present when it extends upward involving the epiglottis and contiguous tissues. Erosion and ulceration appear early, but pain is rarely present. The sputum is tinged with blood, although hemorrhage is not so common as in sarcoma of the nose. The tumors are usually small in size when recognized, as they interfere with respiration before becoming large. The growth does not extend to the adjacent tissues, but if it begins in them it often extends into the larynx. The author recently saw a case in which the posterior wall of the pharynx and larynx were involved, the growth being about the size of a large filbert. A cachexia sometimes develops, and is due to obstructed respiration.

Diagnosis: The tumor is soft and grumous, with an irregularly rounded outline, and is of a pinkish-gray or purplish-gray color. A portion should be removed and submitted to microscopical examination.

The **prognosis** is much more grave than that of sarcoma of the nose. It is almost as fatal as carcinoma of the larynx. The average length of life is less than two years.

Sarcoma of the larynx—treatment: Extirpation is sometimes followed by a cure, although this is rare. Complete resection or extirpation of the entire larynx is usually indicated, as a less extensive operation will be of but temporary value. A preliminary tracheotomy should be performed before removing the larynx, as the anæsthetic must be administered through the artificial opening.

Carcinoma of the Larynx.

Etiology: Heredity exerts an important influence in about 25 per cent. of the cases. It is more frequent in males, and occurs late in life.

Carcinoma of the larynx—symptoms: These will depend upon the location, size, and stage of development of the cancer. If it is located upon the cords or ventricular bands, the voice will be affected. If the posterior commissure is affected, there will be cough; while if located on the anterior wall, the function of the epiglottis will be impaired, thus giving rise to dysphagia. If it is in the ulcerative stage, there will be a foul odor attended by cough, while pain will be more pronounced. At first the voice is weakened and changed in quality or pitch; after a time it is entirely lost. Hemorrhage may occur after the ulceration has formed. It is not usually a fatal or dangerous complication. The cancerous cachexia may or may not be present in the early and middle stages of the disease.

Diagnosis: This must be based upon the location of the tumor, the character and extent of the ulceration, the existence of pain after a somewhat delayed period, and upon a microscopical examination of a piece of the diseased tissue. This must be removed from a non-ulcerated portion of the growth, as the granular surface of the ulcer will not show characteristic *inward proliferation of the epithelium.* The growth is nearly always supraglottic.

Carcinoma of the larynx—treatment: Total extirpation of the larynx is followed by about 10 per cent. of recoveries, and offers the best mode of treatment now known. Less radical measures are attended by less favorable results.

Palliative remedies should be used locally to mitigate the distressing symptoms. The secretion can be removed by gargles containing 10 per cent. of carbonate of sodium, after which hydrozone in 20 per cent. solution should be used. The alkaline solution should be used first, as the hydrozone coagulates the albumin in the secretion and renders it difficult of removal. Powders containing morphine or cocaine, or both combined, afford relief to the sharp, lancinating pains. The fetor may be removed or modified by solutions containing 2 per cent. of permanganate of potassium or an equal amount of carbolic acid.

PRESCRIPTIONS FOR EAR, NOSE, AND THROAT.

The following prescriptions have been selected from the text-books mentioned in the preface and from those used by the author. Some of them have appeared in the text, but most of them are additional remedies which have been found to be valuable. They are classified under the diseases in which they are most useful.

Simple Cleansing Solutions:

℞. Sod. bicarb.,
Sod. boratis, sod. chloridi, āā ℥j.—M.

Sig.—Add a teaspoonful to a pint of lukewarm water and use with a syringe or atomizer to cleanse the nose from thick mucus and crusts.

Simple Cleansing Solutions—*Continued:*

℞. Sod. bicarb.,	gr. xv.
Sod. bibor.,	gr. xv.
Acid. carbol.,	gr. iv.
Glycerin.,	♏xlv.
Aquæ,	ad ℥j.—M.

Sig.—Dobell's solution. Dilute with equal parts of water and use with atomizer; or snuff up the nose.

℞. Acidi acetici,	ʒiiss.
Glycerin.,	ʒiij.
Aquæ,	ad ℥x.—M.

Sig.—To be used as an antiseptic and stimulating wash in the nose and nasopharynx in the course of the exanthematous fevers.

Hay Fever, or Hyperæsthetic Rhinitis:

℞. Acidi carbolici,	gr. xxx.
Ammonii carbonatis,	℥j.
Pulv. carbonis ligni,	℥j.
Olei lavandulæ,	♏xx.
Tinct. benzoin. co.,	℥ss.—M.

Sig.—Uncork and inhale to relieve the congested condition of the mucous membrane in acute coryza and hay fever.

℞. Acidi carbolici,	gr. xxx.
Ext. pini canadensis dest.,	♏xx.
Liq. vaselin.,	℥j.—M.

Sig.—To be used in the acute stage of hay fever for the anæsthetic effect of the carbolic acid.

(INGALS.)

℞. Zinci valerianatis,	gr. j.
Pil. asafœtid. co.,	gr. ij.—M.

Ft. pil. i.

Sig.—Give one or two pills two or three times a day to relieve the neurasthenic symptoms occurring in hay fever.

(SIR MORELL MACKENZIE.)

℞. Glycerit. acid. carbol.,	℥j.
Quininæ hydrochlor.,	ʒj.—M.

Sig.—To be dissolved with the aid of heat and one two-thousandths part of perchloride of mercury added. To be applied with caution to the nasal mucous membrane every two or three days for the relief of the acute symptoms of hay fever.

(Sir Andrew Clark.)

℞. Acid. chromici cryst.,	gr. $\frac{1}{6}$, $\frac{1}{8}$, $\frac{1}{4}$.
Aquæ dest.	℥j.—M.

Ft. nebulæ and use to check sneezing.

Chronic Rhinitis:

℞. Potass. chlor.,	gr. x–xx.
Aquæ,	℥j.—M.

Sig.—To be used as a spray in rhinitis with collapse.

℞. Eucalyptol,	♏xv.
Menthol,	gr. xv.
Camphor.,	gr. lx.
Ol. pini compilonis,	ʒss.
Ol. rosæ	♏ij.
Liq. vaselin.,	q. s. ad ℥ij.—M.

Sig.—Use in the nose with an atomizer in mild cases of hypertrophic rhinitis.

℞. Ichthyol,	gr. xlviij.
Lanolin,	
Vaselin.,	āā ʒiv.—M.

Sig.—Use on a cotton-wound applicator to massage the nasal mucous membrane in chronic forms of rhinitis.

Ozæna and Atrophic Rhinitis:

℞. Hydrarg. ammoniati,	gr. iv.
Pulv. sacch. alb.,	℥ss.—M.

Sig.—Insufflate into the nose to stimulate the mucous membrane in ozæna.

Ozæna and Atrophic Rhinitis—*Continued:*

℞. Hydrarg. oxidi rubr.,	gr. iv.
Pulv. sacch. alb.	℥ss.—M.

Sig.—To be blown into the nose after cleansing in ozæna.

℞. Iodoformi,	gr. v–x.
Menthol,	gr. iij–v.
Lanolin,	℥ss.
Liq. vaselin.,	℥ss.—M.

Ft. ung.

Sig.—To be applied to the interior of the nose with a brush in ozæna or ulcer of the septum.

℞. Creolin,	♏j–iv.
Aquæ,	℥j.—M.

Sig.—Antiseptic and deodorant, to be used in atrophic rhinitis, syphilitic and other ulcerations, and in diseases of the accessory sinuses.

℞. Sanguinariæ canadens.,	♏v–xxx.
Aquæ tepid.,	Oss.—M.

Ft. lotio.

Sig.—To be used with an atomizer or syringe night and morning for ozæna.

℞. Zinc sozoiodol,	gr. xlviij.
Talc.,	q. s. ℥j.—M.

Sig.—To be blown into the nose in atrophic rhinitis after cleansing the nose from crusts.

Diphtheria and Pseudodiphtheria:

℞. Menthol,	gr. xlviij.
Toluol,	ʒij.
Sol. of perchloride of iron (fort.),	gr. xx.
Alcohol absol.,	q. s. ad ℥j.—M.

Sig.—To be applied locally in diphtheria or pseudodiphtheria.

(LOFFLER.)

℞. Acidi lactici, ʒj–ij.
Aquæ, ℥j.—M.

Sig.—To be used with an atomizer in diphtheria as a solvent of the membranous exudate.

(L. Browne.)

℞. Acidi sulphurosi, ℥ss–j.
Aquæ, ad ℥x.—M.

Sig.—To be used as a gargle in diphtheria, pseudodiphtheria, and in mycosis. By some it is considered as almost specific in diphtheria.

Empyema of the Accessory Sinuses:

℞. Aristol,

℞. Europhen,

℞. Nosophen,

℞. Iodol,

℞. Iodoform.

Sig.—To be blown into the nares in empyema of the accessory sinuses.

Relaxed Uvula:

℞. Aluminis,
Acidi tannici, āā gr. lx.
Aquæ rosæ, ℥x.—M.

Sig.—To be used as a gargle in relaxation of the uvula and congestion of the fauces.

Hæmostatics:

℞. Antipyrin, gr. xv.
Aquæ, ad ℥j.—M.

Sig.—To be used in the nose and throat as a hæmostatic.

℞. Pulv. fol. matico,
Pulv. amyl. exsic., āā equal parts.—M.

Sig.—To be insufflated into the nose for epistaxis.

Adenoids:

℞. Glycerit. acid. tannici,	ʒiij.
Aquæ,	ad ℥iij.—M.

Sig.—One teaspoonful to a wineglassful of warm water, to be injected down the nose night and morning, as a remedy for postnasal adenoids.

Dry Catarrh of the Nose, Pharynx, and Larynx:

℞. Iodini,	gr. v–xx.
Potassii iodidi,	gr. x–xxx.
Ol. gaulther. (ol. menth. pip.),	♏v.
Glycerin.,	℥j.—M.

Make four solutions, varying the amount of iodine and potassium iodide between the limits given in the prescription.

Sig.—In dry rhinitis, pharyngitis, and laryngitis begin with the weakest solution and gradually increase to the strongest to stimulate the glandular function of the mucous membrane.

Acute Tonsillitis and Pharyngitis:

℞. Guaiacol,	
Olive oil,	āā ʒiv.—M.

Sig.—Apply to the fauces of pharynx in acute tonsillitis and pharyngitis.

℞. Argent. nitrat.,	
Aquæ dest.,	āā ʒiv.—M.

Sig.—It may be used instead of the guaiacol mixture. One or two applications of the above mixture are often sufficient to arrest acute inflammations of the throat and pharynx if applied in the first stage.

℞. Red gum lozenges (Wyeth).

Sig.—To be dissolved in the mouth at frequent intervals in mild attacks of acute pharyngitis.

℞. Guaiacol,	℥ss.
Ol. amygdalæ dulcis,	℥ss.—M.

Sig.—To be applied with a cotton-wound probe in acute inflammations of the throat.

It is also applied to relieve pain.

Laryngitis :

℞. Tr. iod.,	ʒj.
Glycerin.,	℥ss.
Aquæ,	ad ℥iij.—M.

Sig.—To be used to moisten compress in laryngitis.

℞. Ammonii carbonatis,	gr. ij–iv.
Tr. scillæ,	♏x.
Tr. digitalis,	♏v.
Liq. strychninæ,	♏ij.
Infus. cascarillæ,	ad ℥j.—M.

Sig.—Give one teaspoonful every three to six hours as an expectorant in subacute catarrh of the larynx.

℞. Eucalyptol,	♏x.
Menthol,	gr. v.
Camphor.,	gr. v.
Liq. vaselin.,	q. s. ad ℥j.—M.

Sig.—To be used in acute laryngitis after cleansing the throat with aqueous solutions.

Tubercular Laryngitis :

℞. Menthol,	gr. xlviij.
Ol. oliv.,	q. s. to make ℥j.—M.

Sig.—To be applied to the larynx in laryngeal tuberculosis with a cotton-wound applicator. or an atomizer.

(Rosenberg.)

℞. Ol. eucalyptol,	ʒij.
Ol. terebinth.,	ʒj.
Magnesiæ carb. levis,	ʒij.
Aquæ,	q. s. ad ℥iij.—M.

Sig.—A teaspoonful in a pint of hot water. Inhale the vapor to loosen the secretion and allay the cough in tubercular laryngitis.

Nervous Dyspepsia of Singers:

R. Quininæ sulphatis,	
Acidi carbolici,	
Extract. krameriæ,	āā gr. ss.
Pep.,	gr. ij.—M.

Ft. pilula.

Sig.—Take one before each meal at which meat is taken in cases where there is sluggish digestion with flatulency.

This is of special value for vocalists, actors, and speakers, in whom the digestive system is frequently impaired by nervousness.

Syphilis:

℞. Potassii chloratis,	gr. xc–cxx.
Glycerin.,	ʒij.
Aquæ,	ad ℥x.—M.

Sig.—To be used as a mouth-wash in syphilis during the administration of mercury.

R. Liq. hydrarg. nitratis,	ʒj–iv.
Aquæ,	q. s. ad ℥j.—M.

Sig.—To be applied to the sloughing ulcer of tertiary syphilis. As the application is very painful, cocaine should first be applied.

Inspissated Cerumen:

℞. Sodii bicarb.,	gr. x.
Glycerin.,	ʒj.
Aquæ,	ad ʒiv.—M.

Sig.—Drop into ear three times daily to soften inspissated cerumen preparatory to removing with a syringe.

℞. Hydrozone (15 vol.),	
Aquæ,	āā ʒiij.—M.

Sig.—Same as preceding.

Eczema of the Auricle and Meatus:

℞. Acidi carbolici,	gr. v.
Spirit. rectif.,	ʒij.
Glycerin.,	ʒij.
Aquæ,	ʒiv.—M.

Sig.—Paint external meatus in scaly eczema with pruritus.

℞. Liniment. calcis,	
Ung. hydr. nitratis,	āā ʒiss.
Liq. carbonis detergent.,	♏xij.
Ung. zinci oxid.,	ad ℥j.—M.

Sig.—For external use in scaly eczema of the auricle or external auditory canal.

R. Liquor plumbi subacet.,	ʒj.
Glycerin.,	ʒij.
Aquæ,	ad ℥iv.—M.

Sig.—In eczema or diffused inflammation of the auricle and meatus the parts should be frequently sponged with the lotion.

Furunculosis of the External Meatus:

℞. Menthol,	gr. ij.
Iodoformi,	gr. iv.
Lanolin,	ʒij.
Vaselin.,	ʒij.—M.

Sig.—Apply on cotton plug in furunculosis of the external meatus.

℞. Acidi carbolici,	gr. xx.
Glycerin.,	
Aquæ,	āā ʒiv.—M.

Sig.—Local application to relieve pruritus.

Mastoid Periostitis:

℞. Tinct. iodi.

Sig.—In mild cases of mastoid periostitis and in disease of the labyrinth; external use.

Anodyne Mixtures :

℞. Bismuthi subnit.,	
Pulv. acaciæ,	āā ʒij.
Iodoformi,	ʒss.
Morph. sulph.,	gr. xx.
Acidi tannici,	ʒss.—M.

Sig.—Insufflate into the larynx half an hour before taking food in cases of painful deglutition in tuberculosis and malignant disease.

The iodoform may be omitted if the odor is disagreeable to the patient.

℞. Cocain.,	gr. xx.
Iodoformi,	ʒij.
Zinc. stearat.,	ʒj.—M.

Sig.—Blow into the throat or nose for the relief of pain in tubercular or malignant ulcers.

℞. Camphoræ (reduced to fine powder with a few drops of rectified spirits),	
Chloral hydrat.,	āā ℥ss.—M.

Sig.—To be applied externally as an anæsthetic in neuralgic and other painful affections of the throat.

℞. Acidi carbolici (1 to 5 per cent.),	
Glycerin.,	āā ℥j.—M.

Sig.—Drop into ear to relieve pain in middle-ear inflammation.

Suppurative Otitis Media :

℞. Acidi carbolici,	gr. xx.
Sp. vini rectif.,	
Aquæ,	āā ℥j.—M.

Sig.—Instil into ear after drying and cleansing in chronic purulent inflammation of the middle ear.

℞. Iodoformi,	ʒss.
Sp. vini rectif,	℥j.—M.

Sig.—Instil in chronic purulent inflammation of the middle ear.

Tinnitus Aurium :

Infus. digitalis,	℥iv.—M.

Sig.—Two teaspoonfuls twice daily in pulsating forms of tinnitus.

℞. Sodii brom.,	gr. xv–xxx.

Sig.—Dissolve in a tumbler of water and take at bedtime ; in cerebral symptoms and vertigo occurring in the course of ear disease.

℞. Ammonii chloridi,	gr. xx.
Sodii iodidi,	gr. iij.
Ext. glycyrrh. liq.,	♏xx.
Aquæ,	ad ℥j.—M.

Sig.—To be used internally in chronic nasopharyngitis associated with middle-ear inflammation and tinnitus. Preferably given at 11 A. M. and 4 P. M.

℞. Potassii iodidi,	gr. xxx.
Camphoræ,	gr. vj.
Ol. menth. pip.,	♏v.
Ung. simpl.,	ʒiv.—M.

Sig.—To be applied with friction to the mastoid surface for the relief of tinnitus aurium.

(GRUBER.)

℞. Canthos (Johnson & Johnson).

Sig.—To produce vesication over the mastoid in facial paralysis, in labyrinthine disease, in paralysis of the auditory nerve, otalgia, and in distressing forms of tinnitus aurium.

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